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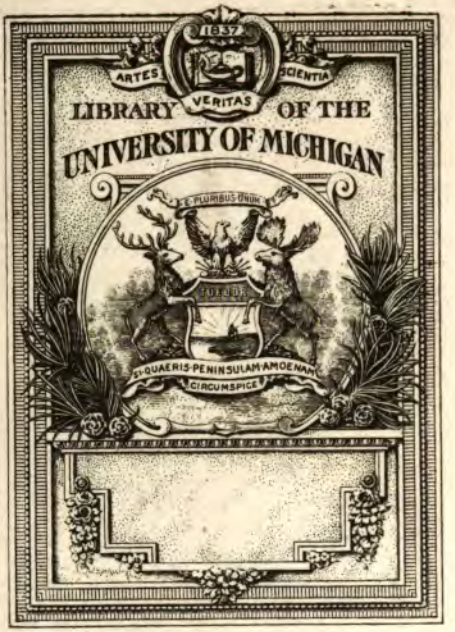

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MEDICO-CHIRURGICAL TRANSACTIONS

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PRESIDENTS OF THE SOCIETY FROM ITS FORMATION, AS THE MEDICO-CHIRURGICAL SOCIETY, 1805.

ELECTED

1805. WILLIAM SAUNDERS, M.D.
1808. MATTHEW BAILLIE, M.D.
1810. SIR HENRY HALFORD, BART., M.D., G.C.H.
1813. SIR GILBERT BLANE, BART., M.D.
1815. HENRY CLINE
1817. WILLIAM BABINGTON, M.D.
1819. SIR ASTLEY PASTON COOPER, BART., K.C.H., D.C.L.
1821. JOHN COOKE, M.D.
1823. JOHN ABERNETHY
1825. GEORGE BIRKBECK, M.D.
1827. BENJAMIN TRAVERS
1829. PETER MARK ROGET, M.D.
1831. SIR WILLIAM LAWRENCE, BART.
1833. JOHN ELLIOTSON, M.D. (First President of the Society after its Incorporation as the Royal Medical and Chirurgical Society, 1834).
1835. HENRY EARLE
1837. RICHARD BRIGHT, M.D., D.C.L.
1839. SIR BENJAMIN COLLINS BRODIE, BART., D.C.L.
1841. ROBERT WILLIAMS, M.D.
1843. EDWARD STANLEY
1845. WILLIAM FREDERICK CHAMBERS, M.D., K.C.H.
1847. JAMES MONCRIEFF ARNOTT
1849. THOMAS ADDISON, M.D.
1851. JOSEPH HODGSON
1853. JAMES COPLAND, M.D.
1855. CÆSAR HENRY HAWKINS
1857. SIR CHARLES LOCOCK, BART., M.D.
1859. FREDERIC CARPENTER SKEY
1861. BENJAMIN GUY BABINGTON, M.D.
1863. RICHARD PARTRIDGE
1865. SIR JAMES ALDERSON, M.D., D.C.L.
1867. SAMUEL SOLLY
1869. SIR GEORGE BURROWS, BART., M.D., D.C.L.
1871. THOMAS BLIZARD CURLING
1873. CHARLES JAMES BLASIUS WILLIAMS, M.D.
1875. SIR JAMES PAGET, BART., D.C.L., LL.D.
1877. CHARLES WEST, M.D.
1879. JOHN ERIC ERICHSEN
1881. ANDREW WHYTE BARCLAY, M.D.
1882. JOHN MARSHALL
1884. SIR GEORGE JOHNSON, M.D.
1886. GEORGE DAVID POLLOCK
1888. SIR EDWARD HENRY SIEVEKING, M.D., LL.D.
1890. TIMOTHY HOLMES
1892. SIR ANDREW CLARK, BART., M.D., LL.D., F.R.S. (*Sir Andrew Clark died 6th November*, 1893, *and Dr. W. S. Church, Senior Vice-President, officiated as President until the following 1st March*, 1894.)
1894. JONATHAN HUTCHINSON, F.R.S.

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HONORARY FELLOWS.

(Limited to Twelve.)

Elected

1887 FLOWER, SIR WILLIAM HENRY, K.C.B., LL.D., F.R.S., Director of the Natural History Department, British Museum, Cromwell road.

1887 FOSTER, MICHAEL, M.D., LL.D., F.R.S., Professor of Physiology in the University of Cambridge.

1883 FRANKLAND, EDWARD, M.D., D.C.L., Ph.D., F.R.S., Corresponding Member of the Academy of Sciences of France; The Yews, Reigate Hill, Reigate.

1868 HOOKER, SIR JOSEPH DALTON, C.B., M.D., K.C.S.I., D.C.L., LL.D., F.R.S., Corresponding Member of the Academy of Sciences of France; The Camp, Sunningdale.

1868 HUXLEY, the Right Hon. THOMAS HENRY, D.C.L., LL.D., F.R.S., Corresponding Member of the Academies of Sciences of St. Petersburg, Berlin, and Dresden.

1878 LUBBOCK, the Right Hon. SIR JOHN, Bart., M.P., D.C.L., LL.D., F.R.S., High Elms, Farnborough, Kent, R.S.O.

1873 STOKES, SIR GEORGE GABRIEL, Bart., M.A., D.C.L., LL.D., F.R.S., M.P., Lucasian Professor of Mathematics in the University of Cambridge; Lensfield Cottage, Cambridge.

1887 TURNER, SIR WILLIAM, M.B., D.C.L., LL.D., F.R.S., Professor of Anatomy in the University of Edinburgh; 6, Eton Terrace, Edinburgh.

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FOREIGN HONORARY FELLOWS.

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1878 Baccelli, Guido, M.D., Professor of Medicine at Rome.

1887 Billings, John S., M.D., D.C.L.Oxon., Surgeon U.S.Army; Librarian, Surgeon-General's Office, Washington.

1883 DuBois Reymond, Emil, M.D., Professor of Physiology, Berlin; N. W. Neue Wilhelmstrasse 15, Berlin.

1887 von Esmarch, Friedrich, M.D., Professor of Surgery in the University of Kiel.

1868 Kölliker, Albert, Professor of Anatomy in the University of Würzburg.

1868 Larrey, Hippolyte Baron, Member of the Institute of France; Inspector of the "Service de Santé Militaire," and Member of the "Conseil de Santé des Armées;" Commander of the Legion of Honour, &c.; Rue de Lille, 91, Paris.

1883 Pasteur, Louis, LL.D., Member of the Institute of France.

1878 von Scanzoni, Friedreich Wilhelm, Royal Bavarian Privy Councillor; Professor of Midwifery in the University of Würzburg.

1856 von Virchow, Rudolph, M.D., LL.D., Professor of Pathological Anatomy in the University of Berlin; Corresponding Member of the Academy of Sciences of France; 10, Schellingstrasse, Berlin.

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FELLOWS

OF THE

ROYAL MEDICAL AND CHIRURGICAL SOCIETY

OF LONDON.

EXPLANATION OF THE ABBREVIATIONS.

P.—President.	C.—Member of Council.
V.P.—Vice-President.	*Sci. Com.*—A Scientific Committee.
T.—Treasurer.	*Ho. Com.*—House Committee.
L.—Hon. Librarian.	*Lib. Com.*—Library Committee.
S.—Hon. Secretary.	*Bldg. Com.*—Building Committee.

The abbreviations *Trans.* and *Pro.*, followed by figures, show the number of Papers which have been contributed to the 'Transactions' or 'Proceedings' by the Fellow whose name they follow. *Referee, Sci. Com.*, and *Lib. Com.*, *Bldg. Com.*, *Ho. Com.*, with the dates of office, are attached to the names of those who have served as Referees of papers and on the Committees of the Society.

Those marked thus (†) have paid the Composition Fee in lieu of further annual subscriptions.

Those marked thus (*) have paid the Composition Fee entitling them to receive the 'Transactions.'

RESIDENT FELLOWS.

Elected

†1877 ABERCROMBIE, JOHN, M.D., Physician to, and Lecturer on Forensic Medicine at, Charing Cross Hospital; 23, Upper Wimpole street, Cavendish square. *Trans.* 1.

1885 ABRAHAM, PHINEAS S., M.A., M.D., Lecturer on Physiology and Histology at the Westminster Hospital; 2, Henrietta street, Cavendish square.

1885 ACLAND, THEODORE DYKE, M.D., Assistant Physician to St. Thomas's Hospital and Physician to the Hospital for Consumption and Diseases of the Chest, Brompton; 74, Brook street, Grosvenor square, W.

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†1852 ADAMS, WILLIAM, Consulting Surgeon to the Great Northern Central Hospital, the National Hospital for the Paralysed and Epileptic, and the National Orthopædic Hospital; 5, Henrietta street, Cavendish square. C. 1873-4. *Trans.* 3.

1867 AIKIN, CHARLES ARTHUR, 12, Ladbroke terrace, Notting hill.

1879 ALLCHIN, WILLIAM HENRY, M.D., F.R.S. Ed., Physician to the Westminster Hospital; 5, Chandos street, Cavendish square.

1890 ALLINGHAM, HERBERT WILLIAM, 25, Grosvenor street, Grosvenor square.

†1863 ALTHAUS, JULIUS, M.D., Senior Physician to the Hospital for Epilepsy and Paralysis, Regent's Park; 48, Harley street, Cavendish square. *Trans.* 2.

1888 ANDERSON, JOHN, M.D., C.I.E., Physician to the Seamen's Hospital, Greenwich; 9, Harley street, Cavendish square.

1890 ANDERSON, WILLIAM, Surgeon to St. Thomas's Hospital; Professor of Anatomy to the Royal Academy of Arts; 2, Harley street, Cavendish square.

1891 ANDREWES, FREDERICK WILLIAM, M.B., 35, Welbeck street, Cavendish square.

1891 ANDREWS, LAUNCELOT, M.B., 22, Cheyne gardens, Manor street, Chelsea.

1888 ARKLE, CHARLES, M.D., Pathologist to Charing Cross Hospital; 66, Wimpole street, Cavendish square.

1893 BAILEY, ROBERT COZENS, M.S., 2, Museum Chambers, Bury street, Bloomsbury.

1865 BAKER, WILLIAM MORRANT, Consulting Surgeon to the Evelina Hospital for Sick Children; 39, Woburn square. C. 1878-9. V.P. 1889. *Referee*, 1886-8. *Lib. Com.* 1876-7. *Trans.* 7.

1887 BALL, JAMES BARRY, M.D., Physician to the West London Hospital; 12, Upper Wimpole street, Cavendish square.

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1885 BALLANCE, CHARLES ALFRED, M.S., Assistant Surgeon to St. Thomas's Hospital and to the Hospital for Sick Children, Great Ormond street; Surgeon to the National Hospital for the Paralysed and Epileptic, Queen square; 106, Harley street, Cavendish square. *Trans.* 1.

1879 BARKER, ARTHUR EDWARD JAMES, Professor of the Principles and Practice of Surgery and Professor of Clinical Surgery at University College, and Surgeon to University College Hospital, London; 87, Harley street, Cavendish square. *Trans.* 7.

†1876 BARLOW, THOMAS, M.D., B.S., *Trustee for Debenture-holders*, Physician to University College Hospital, and to the Hospital for Sick Children, Great Ormond street; 10, Wimpole street, Cavendish square. C. 1892. *Trans.* 2.

1893 BARRETT, HOWARD, 49, Gordon square.

1880 BARROW, A. BOYCE, Assistant Surgeon to King's College Hospital; 37, Wimpole street, Cavendish square.

1859 BARWELL, RICHARD, Consulting Surgeon to the Charing Cross Hospital; 55, Wimpole street. C. 1876-77. V.P. 1883-4. *Referee*, 1868-75, 1879-82. *Trans.* 12.

†1868 BASTIAN, HENRY CHARLTON, M.A., M.D., F.R.S., Professor of Medicine in University College, London; Physician to University College Hospital and to the National Hospital for the Paralysed and Epileptic; 8A, Manchester square. C. 1885. *Referee*, 1886—. *Trans.* 2.

1890 BATEMAN, WILLIAM A. F., Bridge House, Richmond, Surrey.

1891 BATTEN, FREDERICK E., M.A., M.B., B.C.Cantab., 15, Airlie gardens, Campden hill, Kensington.

1875 BEACH, FLETCHER, M.B., Winchester House, Kingston Hill [64, Welbeck street, W.].

1883 BEALE, EDWIN CLIFFORD, M.A., M.B., Physician to the City of London Hospital for Diseases of the Chest, and Physician to the Great Northern Central Hospital; 23, Upper Berkeley street.

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1862 BEALE, LIONEL SMITH, M.B., F.R.S., Professor of the Principles and Practice of Medicine in King's College, London, and Physician to King's College Hospital; 61, Grosvenor street. C. 1876-7. *Referee*, 1873-5. *Trans.* 1.

1880 BEEVOR, CHARLES EDWARD, M.D., Physician for Out-patients to the National Hospital for the Paralysed and Epileptic, and to the Great Northern Hospital; 33, Harley street, Cavendish square. *Trans.* 1.

1880 BENNETT, ALEX. HUGHES, M.D., Physician to the Westminster Hospital; 76, Wimpole street, Cavendish square. *Trans.* 1.

1883 BENNETT, STORER, Dental Surgeon to, and Lecturer on Dental Surgery at, the Middlesex Hospital; Dental Surgeon to, and Lecturer on Dental Surgery and Pathology at, the Dental Hospital of London; 17, George street, Hanover square.

1877 BENNETT, WILLIAM HENRY, Surgeon to St. George's Hospital; 1, Chesterfield street, Mayfair. C. 1893-4. *Referee*, 1892-93. *Trans.* 4.

†1845 BERRY, EDWARD UNWIN, 17, Sherriff road, West Hampstead.

1885 BERRY, JAMES, B.S., Demonstrator of Anatomy, St. Bartholomew's Hospital; Surgeon to, and Lecturer on Clinical Surgery at, the Royal Free Hospital; 60, Welbeck street, Cavendish square.

1893 BIDWELL, LEONARD A., Senior Assistant Surgeon to the West London Hospital; 59, Wimpole street.

†1856 BIRD, WILLIAM, Consulting Surgeon to the West London Hospital; Bute House, Hammersmith.

†1851 BIRKETT, JOHN, F.L.S., Consulting Surgeon to Guy's Hospital; Corresponding Member of the Société de Chirurgie of Paris; Inspector of Anatomy for the Provinces in England and Wales; 62, Green street, Grosvenor square. L. 1856-7. S. 1863-5. C. 1867-8. T. 1870-78. V.P. 1879-80. *Referee*, 1851-5, 1866, 1869. *Sci. Com.* 1863. *Lib. Com.* 1852. *Trans.* 8.

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1881 Biss, Cecil Yates, M.D., Senior Assistant Physician to, and Lecturer on Pharmacology and Therapeutics at, the Middlesex Hospital, and Physician to the Hospital for Consumption, Brompton; 135, Harley street, Cavendish square. *Trans.* 2.

1865 Blandford, George Fielding, M.D., late Lecturer on Psychological Medicine at St. George's Hospital; 48, Wimpole street, Cavendish square. C. 1883-4.

1891 Bokenham, Thomas Jessopp, 10, Devonshire street, Portland place.

†1846 Bostock, John Ashton, C.B., *Hon. Treasurer;* Hon. Surgeon to H.M. the Queen; Deputy Surgeon-General; 73, Onslow gardens, Brompton. C. 1861-2. V.P. 1870-71. T. 1888—. *Sci. Com.* 1867. *Ho. Com.* 1894—.

1890 Bostock, R. Ashton, Surgeon, Scots Guards, 73, Onslow gardens, Brompton.

1882 Bowlby, Anthony Alfred, Assistant Surgeon to St. Bartholomew's Hospital; 24, Manchester square. *Trans.* 5.

*1870 Bowles, Robert Leamon, M.D., 16, Upper Brook street, Grosvenor square. *Trans.* 1.

1893 Bowman, Henry Moore, M.D., 2, Museum chambers, Bury street, W.C.

1886 Boxall, Robert, M.D., Assistant Obstetric Physician to, and Lecturer on Practical Midwifery at, the Middlesex Hospital; 29, Weymouth street, Portland place.

1884 Boyd, Stanley, M.B., Surgeon to, and Lecturer on Anatomy at, Charing Cross Hospital; Surgeon to the Paddington Green Children's Hospital; 134, Harley street, Cavendish square. *Trans.* 1.

1890 Bradford, John Rose, M.D., D.Sc., F.R.S., Assistant Physician to University College Hospital; 52, Upper Berkeley street, Portman square.

1883 Bradshaw, James Dixon, M.B., Savile Club, Piccadilly, W.

1890 Brinton, Roland Danvers, M.D., 8, Queen's Gate terrace.

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1868 BROADBENT, SIR WILLIAM HENRY, Bart., M.D., Physician to, and Lecturer on Clinical Medicine at, St. Mary's Hospital; Consulting Physician to the London Fever Hospital; Physician in Ordinary to H.R.H. the Prince of Wales; 84, Brook street, Grosvenor square. C. 1885. *Referee*, 1881-4, 1891—. *Trans.* 5.

†1851 BRODHURST, BERNARD EDWARD, Surgeon to the Royal Orthopædic Hospital and to the Royal Hospital for Incurables; Consulting Surgeon, Belgrave Hospital for Children; Corresponding Member Société de Chirurgie, Paris, and of the Academy of Sciences, Rome; 20, Grosvenor street. C. 1868-9. *Lib. Com.* 1862-3. *Trans.* 2. *Pro.* 1.

1872 BRODIE, GEORGE BERNARD, M.D., Consulting Physician-Accoucheur to Queen Charlotte's Hospital; 8, Chesterfield street, Mayfair. *Trans.* 1.

1891 BRODIE, CHARLES GORDON, Senior Demonstrator of Anatomy, Middlesex Hospital Medical School; Assistant Surgeon, North-West London Hospital; 30, Harley street, Cavendish square.

1880 BROWNE, JAMES WILLIAM, M.B., 7, Norland place, Holland Park.

1881 BROWNE, OSWALD AUCHINLECK, M.A., M.B., Physician to the Royal Hospital for Diseases of the Chest; 43, Bedford square.

1874 BRUCE, JOHN MITCHELL, M.D., *Hon. Secretary*; Physician to, and Lecturer on Medicine at, the Charing Cross Hospital; Physician to the Hospital for Consumption, Brompton; 70, Harley street. C. 1892. S. 1894—. *Sci. Com.* 1889—. *Referee*, 1886-91. *Lib. Com.* 1888-91. *Trans.* 3.

1871 BRUNTON, THOMAS LAUDER, M.D., D.Sc., LL.D., F.R.S., Assistant Physician to, and Lecturer on Materia Medica and Therapeutics at, St. Bartholomew's Hospital; 10, Stratford place, Oxford street. C. 1888-9. *Referee*, 1880-87. *Lib. Com.* 1882-7. *Trans.* 1.

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1860 BRYANT, THOMAS, Consulting Surgeon to Guy's Hospital; Member of the Société de Chirurgie, Paris; 65, Grosvenor street, Grosvenor square. C. 1873-4. V. P. 1885-6. *Sci. Com.* 1863. *Referee*, 1882-4. *Lib. Com.* 1868-71. *Trans.* 15. *Pro.* 1.

1864 BUCHANAN, SIR GEORGE, M.D., F.R.S., late Medical Officer of the Local Government Board; Member of the Senate of the University of London; 27, Woburn square.

1889 BULL, WILLIAM CHARLES, M.B., Aural Surgeon to, and Lecturer on Aural Surgery at, St. George's Hospital; 35, Clarges street, Piccadilly.

1893 BURGHARD, FRÉDÉRIC FRANÇOIS, M.D., M.S.; 46, Weymouth street, Portland place.

1885 BUTLER-SMYTHE, ALBERT CHARLES, Senior Surgeon to the Grosvenor Hospital for Women and Children; 76, Brook street, Grosvenor square.

1873 BUTLIN, HENRY TRENTHAM, Surgeon to St. Bartholomew's Hospital; 82, Harley street, Cavendish square. C. 1887-8. *Referee*, 1893—. *Trans.* 4.

1883 BUXTON, DUDLEY WILMOT, M.D., B.S., Administrator, and Teacher of the Use, of Anæsthetics, in University College Hospital; Anæsthetist to the Hospital for Paralysis and Epilepsy, Queen's square, and to the London Dental Hospital; 82, Mortimer street, Cavendish square.

1868 BUZZARD, THOMAS, M.D., Physician to the National Hospital for the Paralysed and Epileptic; 74, Grosvenor street, Grosvenor square. C. 1885-6. *Referee*, 1887—.

1890 CAGNEY, JAMES, M.A., M.D., in charge of Electrical Department, St. Mary's Hospital; Physician to the Hospital for Epilepsy and Paralysis, Regent's Park; 93, Wimpole street, Cavendish square. *Trans.* 1.

1885 CAHILL, JOHN, 12, Seville street, Lowndes square.

1893 CALEY, HENRY ALBERT, M.D., 24, Upper Berkeley street, Portman square.

1887 CALVERT, JAMES, M.D., 36, Queen Anne street, Cavendish square.

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Elected

1891 CAMPBELL, HENRY JOHNSTONE, M.D., Senior Demonstrator of Biology and Demonstrator of Physiology, Guy's Hospital; Assistant Physician, East London Children's Hospital; 54, Welbeck street.

1888 CARLESS, ALBERT, M.S., Assistant Surgeon to King's College Hospital; 10, Welbeck street.

1875 CARTER, CHARLES HENRY, M.D., Physician to the Hospital for Women, Soho square; 45, Great Cumberland place, Hyde Park.

1853 CARTER, ROBERT BRUDENELL, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. George's Hospital; 31, Harley street, Cavendish square. *Trans.* 1.

1888 CAUTLEY, EDMUND, M.D., B.C., 15, Upper Brook street.

1868 CAVAFY, JOHN, M.D., Physician to St. George's Hospital; 2, Upper Berkeley street, Portman square. C. 1887. *Lib. Com.* 1888—. *Trans.* 1.

1871 CAYLEY, WILLIAM, M.D., Physician to, and Lecturer on the Principles and Practice of Medicine at, the Middlesex Hospital; Consulting Physician to the London Fever Hospital and to the North-Eastern Hospital for Children; 27, Wimpole street, Cavendish square. C. 1888. *Referee*, 1886-7. *Lib. Com.* 1886-7. *Trans.* 2.

1879 CHAMPNEYS, FRANCIS HENRY, M.A., M.D., Physician Accoucheur and Lecturer on Obstetric Medicine at St. Bartholomew's Hospital; 42, Upper Brook street, Grosvenor square. *Referee*, 1891—. *Lib. Com.* 1885—. *Trans.* 7.

1868 CHEADLE, WALTER BUTLER, M.D., *Trustee;* Physician to, and Lecturer on Medicine at, St. Mary's Hospital; Consulting Physician to the Hospital for Sick Children; 19, Portman street, Portman square. S. 1886-8. C. 1890-91. *Sci. Com.* 1889—. *Bldg. Com.* 1889-92. *Referee*, 1885. *Trans.* 1.

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1879 CHEYNE, WILLIAM WATSON, M.B., F.R.S., Surgeon to King's College Hospital, and Professor of Surgery in King's College, London; 75, Harley street, Cavendish square. *Referee*, 1894—. *Lib. Com.* 1886-8, 1891—. *Trans.* 1.

1865 CHOLMELEY, WILLIAM, M.D., Physician to the Great Northern Central Hospital; Worthing. C. 1881-2. *Referee*, 1873-80.

1866 CHURCH, WILLIAM SELBY, M.D., *Hon. Treasurer*, Physician to, and Lecturer on Clinical Medicine at, St. Bartholomew's Hospital; 130, Harley street, Cavendish square. C. 1885-6. V.P. 1892-4. T. 1894—. *Referee*, 1874-81.

1879 CLARK, ANDREW, Surgeon to, and Lecturer on Practical Surgery at, the Middlesex Hospital; 71, Harley street, Cavendish square.

1882 CLARKE, ERNEST, M.D., B.S., Surgeon to the Central London Ophthalmic Hospital; Ophthalmic Surgeon to the Miller Hospital; 112, Harley street.

1890 CLARKE, JAMES JACKSON, M.B., Curator of the Museum and Pathologist to St. Mary's Hospital, 9, Old Cavendish street, W.

†1848 CLARKE, JOHN, M.D., 42, Hertford street, Mayfair. C. 1866.

1881 CLARKE, W. BRUCE, M.B., Assistant Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; Surgeon to the West London Hospital; 46, Harley street, Cavendish square. *Trans.* 1.

†1879 CLUTTON, HENRY HUGH, M.A., M.B., Surgeon to, and Lecturer on Surgery at, St. Thomas's Hospital; Surgeon to the Victoria Hospital for Children; 2, Portland place. *Trans.* 1.

1888 COCK, FREDERICK WILLIAM, M.D., 1, Porchester Houses Porchester square.

1865 COOPER, ALFRED, Consulting Surgeon to the West London Hospital; Senior Surgeon to St. Mark's Hospital, 9, Henrietta street, Cavendish square.

1889 COSENS, CHARLES HENRY, 49, Oxford terrace, Hyde Park.

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1892 COTTERELL, EDWARD, 5, West Halkin street, Belgrave square. *Trans.* 1.

†1860 COUPER, JOHN, Surgeon to the Royal London Ophthalmic Hospital, and Consulting Surgeon to the London Hospital; 80, Grosvenor street. C. 1876. *Referee*, 1882-3.

1877 COUPLAND, SIDNEY, M.D., Physician to, and Joint Lecturer on Practical Medicine at, the Middlesex Hospital; 16, Queen Anne street, Cavendish square. C. 1893—. *Referee*, 1892-3.

†1862 COWELL, GEORGE, Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital; Consulting Surgeon to the Victoria Hospital for Children; 3, Cavendish place, Cavendish square. C. 1882-3.

1868 CRAWFORD, SIR THOMAS, K.C.B., M.D., M.Ch., LL.D., Hon. Surgeon to the Queen; Director-General, Army Medical Department (Retired); 5, St. John's Park, Blackheath. C. 1887.

1878 CRICHTON-BROWNE, SIR JAMES, M.D., LL.D., F.R.S., Lord Chancellor's Visitor in Lunacy; 61, Carlisle place Mansions, Victoria street.

1874 CRIPPS, WILLIAM HARRISON, Assistant Surgeon to St. Bartholomew's Hospital; 2, Stratford place, Oxford street. C. 1890-91. *Trans.* 1.

1882 CROCKER, HENRY RADCLIFFE, M.D., Physician to the Skin Department, University College Hospital; late Physician to the East London Hospital for Children; 121, Harley street, Cavendish square. *Trans.* 3.

1868 CROFT, JOHN, Consulting Surgeon to St. Thomas's Hospital; 6, Mansfield street, Cavendish square. C. 1884. V.P. 1890. *Referee*, 1885-88. *Lib. Com.* 1877-8. *Trans.* 2.

1890 CROWLE, THOMAS HENRY RICKARD, 3, Campden Hill road, Kensington.

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1888 CULLINGWORTH, CHARLES JAMES, M.D., Obstetric Physician and Lecturer on Midwifery at St. Thomas's Hospital; 46, Brook street, Grosvenor square.

1879 CUMBERBATCH, A. ELKIN, Aural Surgeon to St. Bartholomew's Hospital, and to the National Hospital for the Paralysed and Epileptic; 17, Queen Anne street.

1873 CURNOW, JOHN, M.D., Professor of Anatomy in King's College, London, and Physician to King's College Hospital; Senior Physician to the Seamen's Hospital; 11, Wimpole street, Cavendish square. *Referee*, 1884—.

1886 DAKIN, WILLIAM RADFORD, M.D., Obstetric Physician to St. George's Hospital; 57, Welbeck street, Cavendish square.

1872 DALBY, SIR WILLIAM BARTLETT, M.B., Consulting Aural Surgeon to St. George's Hospital; 18, Savile row. *Trans.* 3.

1891 DALTON, NORMAN, M.D., Assistant Surgeon to King's College Hospital; 4, Mansfield street, Cavendish square.

1876 DAVIES-COLLEY, J. NEVILLE C., M.C., Surgeon to, and Lecturer on Surgery at, Guy's Hospital; 36, Harley street, Cavendish square. C. 1892-3. *Referee*, 1890-91. *Trans.* 3.

1893 DAVIS, GEORGE WILLIAM, M.D., B.S., Sunningdale, Sidcup, Kent.

1867 DAY, WILLIAM HENRY, M.D., Physician to the Samaritan Free Hospital for Women and Children; 10, Manchester square.

1889 DEAN, HENRY PERCY, M.S., Assistant Surgeon to the London Hospital; 84, Wimpole street, Cavendish square.

1878 DENT, CLINTON THOMAS, Assistant Surgeon to, and Lecturer on Practical Surgery at, St. George's Hospital; 61, Brook street. C. 1890. *Bldg. Com.* 1890-2. *Referee*, 1892—. *Trans.* 4.

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Elected

1893 DICKINSON, THOMAS VINCENT, M.D., 33, Sloane street, S.W.

†1859 DICKINSON, WILLIAM HOWSHIP, M.D., Consulting Physician to St. George's Hospital, and Consulting Physician to the Hospital for Sick Children; Honorary Fellow of Caius College, Cambridge; 9, Chesterfield street, Mayfair. C. 1874-5. V.P. 1887. *Referee*, 1869-73. 1882-6. *Sci. Com.* 1867, 1879, 1889—. *Trans.* 15.

†1891 DICKINSON, WILLIAM LEE, M.D., Assistant Physician to St. George's Hospital; 9, Chesterfield street, Mayfair.

1889 DODD, HENRY WORK, Assistant Surgeon to the Royal Free Hospital, and to the Royal Westminster Ophthalmic Hospital; Ophthalmic Surgeon to the West-End Hospital for Nervous Diseases; 136, Harley street, Cavendish square.

1888 DONELAN, JAMES, M.B., M.C., 2, Upper Wimpole street, Cavendish square.

1879 DONKIN, HORATIO BRYAN, M.D.Oxon., Physician to the Westminster Hospital; Physician to the East London Hospital for Children; 108, Harley street, Cavendish square.

1877 DORAN, ALBAN HENRY GRIFFITHS, Surgeon to the Samaritan Free Hospital; 9, Granville place, Portman square. C. 1893-94. *Lib. Com.* 1891-3. *Trans.* 4.

1891 DOVE, PERCY W., "Carshalton," Stapleton Hall Road, Stroud Green, N.

1879 DREWITT, F. G. DAWTREY, M.D., Physician to the West London Hospital and to the Victoria Hospital for Children; 2, Manchester square.

†1865 DUCKWORTH, SIR DYCE, M.D., LL.D., Hon. Physician to H.R.H. the Prince of Wales; Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; 11, Grafton street, Bond street. C. 1883-4. *Referee* 1885—. *Trans.* 2.

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1876 DUDLEY, WILLIAM LEWIS, M.D., Physician to the City Dispensary; 149, Cromwell road, South Kensington.

1874 DUFFIN, ALFRED BAYNARD, M.D., Professor of Pathological Anatomy in King's College, London, and Physician to King's College Hospital; 18, Devonshire street, Portland place. C. 1893-94.

1871 DUKE, BENJAMIN, Windmill House, Clapham Common.

1880 DUNBAR, JAMES JOHN MACWHIRTER, M.D., Hedingham House, Clapham Common.

1884 DUNCAN, WILLIAM, M.D., Obstetric Physician to, and Lecturer on Midwifery at, the Middlesex Hospital; 6, Harley street, Cavendish square.

1887 DUNN, HUGH PERCY, Assistant Ophthalmic Surgeon to, and Pathologist at the West London Hospital; 54, Wimpole street, Cavendish square.

1863 DURHAM, ARTHUR EDWARD, F.L.S., Surgeon to, and Lecturer on Clinical Surgery at, Guy's Hospital; 82, Brook street, Grosvenor square. C. 1876-7. V.P. 1887. *Referee*, 1880-1. *Sci. Com.* 1867. *Lib. Com.* 1872-5. *Trans.* 5.

1874 DURHAM, FREDERIC, M.B., Senior Surgeon to the North-West London Hospital; late Surgical Registrar to Guy's Hospital; 82, Brook street, Grosvenor square.

1893 DURHAM, HERBERT EDWARD, M.B., 82, Brook street. *Trans.* 1.

1868 EASTES, GEORGE, M.B.Lond., 35, Gloucester place, Hyde Park. C. 1892-3.

1888 ECCLES, ARTHUR SYMONS, M.B., C.M., 23, Hertford street, Mayfair.

1893 ECCLES, WILLIAM MCADAM, M.D., 10, Welbeck street.

1891 EDDOWES, ALFRED, M.D., 25, Old Burlington street.

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1883 EDMUNDS, WALTER, M.C., 75, Lambeth Palace road, Albert Embankment. *Trans.* 2.

1884 EDWARDS, FREDERICK SWINFORD, Surgeon to the West London Hospital, and to St. Peter's Hospital for Stone; 55, Harley street, Cavendish square.

1891 ELAM, GEORGE, M.B., 96, Manor road, Stoke Newington.

†1842 ERICHSEN, JOHN ERIC, LL.D., F.R.S., M.Ch., Surgeon Extraordinary to H.M. the Queen; President of, and Emeritus Professor of Surgery in, University College, London, and Consulting Surgeon to University College Hospital; 6, Cavendish place, Cavendish square. C. 1855-6. V.P. 1868. P. 1879-80. *Referee*, 1866-8, 1884-89. *Lib. Com.* 1844-7, 1854. *Trans.* 2.

1879 EVE, FREDERIC S., Surgeon to the London Hospital; Surgeon to Out-Patients at the Evelina Hospital for Sick Children; 125, Harley street, Cavendish square. *Trans.* 2.

1877 EWART, WILLIAM, M.D., Physician to St. George's Hospital; 33, Curzon street, Mayfair. *Sci. Com.* 1889—. *Trans.* 1.

1872 FAYRER, SIR JOSEPH, K.C.S.I., LL.D., M.D., F.R.S., Surgeon-General; Honorary Physician to H.M. the Queen, (Military) to H.R.H. the Prince of Wales, and Physician to H.R.H. the Duke of Edinburgh; Physician to the Secretary of State for India in Council; President of the Medical Board at the India Office; 53, Wimpole street, Cavendish square. C. 1888. *Referee*, 1881-7.

1863 FENWICK, SAMUEL, M.D., Physician to the London Hospital; 29, Harley street, Cavendish square. C. 1880. *Referee*, 1882—. *Trans.* 4.

1880 FERRIER, DAVID, M.D., LL.D., F.R.S., Professor of Neuropathology in King's College, London, and Physician to King's College Hospital; Physician for Out-patients to the National Hospital for the Paralysed and Epileptic; 34, Cavendish square. *Referee*, 1891—. *Trans.* 2.

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1889 FIELD, GEORGE P., Aural Surgeon to, and Lecturer on Aural Surgery at, St. Mary's Hospital, and Dean of the Medical School; 34, Wimpole street, Cavendish square.

1866 FITZ-PATRICK, THOMAS, A.M., M.D., 30, Sussex gardens, Hyde Park. C. 1894—.

1891 FLETCHER, HERBERT MORLEY, M.D., 98, Harley street, Cavendish square.

1892 FORSBROOK, WILLIAM HENRY RUSSELL, M.D., 139, Buckingham Palace road.

1882 FOWLER, JAMES KINGSTON, M.A., M.D., Physician to, and Lecturer on Pathological Anatomy at, the Middlesex Hospital, and Physician to the Hospital for Consumption, Brompton; 35, Clarges street, Piccadilly.

1880 FOX, THOMAS COLCOTT, B.A., M.B., Physician for Skin Diseases to the Westminster Hospital, and to the Skin Department of the Paddington Green Hospital for Children; late Physician to the Victoria Hospital for Children; 14, Harley street, Cavendish square. *Trans.* 1.

1884 FULLER, CHARLES CHINNER, 10, St. Andrew's place, Regent's Park.

1883 FULLER, HENRY ROXBURGH, M.D., 45, Curzon street, Mayfair.

†1874 GALABIN, ALFRED LEWIS, M.A., M.D., Obstetric Physician to, and Lecturer on Midwifery and the Diseases of Women at, Guy's Hospital; 49, Wimpole st., Cavendish square. C. 1892. *Referee*, 1882-91. *Lib. Com.* 1883-4. *Trans.* 2.

1883 GALTON, JOHN CHARLES, M.A., F.L.S., 10, Upper Cheyne row, Chelsea.

1865 GANT, FREDERICK JAMES, Consulting Surgeon to the Royal Free Hospital; 16, Connaught square, Hyde Park. C. 1880-81. *Referee*, 1886—. *Lib. Com.* 1882-5. *Trans.* 3.

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†1854 GARROD, SIR ALFRED BARING, M.D., F.R.S., Physician Extraordinary to H.M. the Queen; Consulting Physician to King's College Hospital; 10, Harley street, Cavendish square. C. 1867. V.P. 1880-81. *Referee*, 1855-65. *Trans.* 8.

1886 GARROD, ARCHIBALD EDWARD, M.A., M.D., Assistant Physician to the West London Hospital; 9, Chandos street, Cavendish square. *Sci. Com.* 1889—. *Trans.* 5.

1887 GAY, JOHN, 119, Upper Richmond road, Putney.

1866 GEE, SAMUEL JONES, M.D., *Hon. Librarian, Chairman of Trustees for Debenture-holders*, Physician to St. Bartholomew's Hospital; 31, Upper Brook street, Grosvenor square. C. 1883-4. L. (June) 1887—. *Sci. Com.* 1879. *Bldg. Com.* 1889-92. *Referee*, 1885-7. *Lib. Com.* 1871-6. *Trans.* 1.

1885 GELL, HENRY WILLINGHAM, M.B., 36, Hyde Park square.

1878 GERVIS, HENRY, M.D., Consulting Obstetric Physician to St. Thomas's Hospital; Consulting Physician to the Royal Maternity Charity; 40, Harley street, Cavendish square. *Referee*, 1884—. *Trans.* 1.

1880 GIBBONS, ROBERT ALEXANDER, M.D., Physician to the Grosvenor Hospital for Women and Children; 29, Cadogan place.

1893 GILES, ARTHUR EDWARD, M.D., B.Sc., 57, Queen Anne street.

1893 GILL, RICHARD, 72, Wimpole street.

1877 GODLEE, RICKMAN JOHN, M.S., *Hon. Secretary*, Surgeon to University College Hospital, and Professor of Clinical Surgery in University College, London, and Surgeon to the Hospital for Consumption, Brompton; Consulting Surgeon to the North-Eastern Hospital for Children; 19, Wimpole street, Cavendish square. S. 1892-4. *Referee*, 1886-91. *Trans.* 8.

†1870 GODSON, CLEMENT, M.D., Consulting Physician to the City of London Lying-in Hospital; 9, Grosvenor street, Grosvenor square.

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1886 GOLDING-BIRD, CUTHBERT HILTON, M.B., Senior Assistant Surgeon and Lecturer on Physiology at Guy's Hospital; 12, Queen Anne street, Cavendish square. *Trans.* 1.

1883 GOODHART, JAMES FREDERIC, M.D., Physician to Guy's Hospital; Consulting Physician to the Evelina Hospital for Sick Children; 25, Weymouth street, Portland place. *Lib. Com.* 1893—.

1889 GOODSALL, DAVID HENRY, Surgeon to the Metropolitan Hospital; Surgeon to St. Mark's Hospital; 17, Devonshire place, Upper Wimpole street.

1893 GORDON, WILLIAM, M.B., M.C., 80, Elvaston place, Queen's gate.

1877 GOULD, ALFRED PEARCE, M.S., Assistant Surgeon to the Middlesex Hospital; 10, Queen Anne street, Cavendish square. C. 1892-3. *Ho. Com.* 1891—. *Trans.* 2.

1891 GOW, WILLIAM J., M.D., 13, Upper Wimpole street, Cavendish square.

1873 GOWERS, WILLIAM RICHARD, M.D., F.R.S., Consulting Physician to University College Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 50, Queen Anne street, Cavendish square. C. 1891. *Referee* 1888-90. *Lib. Com.* 1884-6. *Trans.* 7.

†1851 GOWLLAND, PETER YEAMES, Surgeon to St. Mark's Hospital; Surgeon-Major Hon. Artillery Company; 82, Gloucester terrace, Hyde park.

1892 GRANT, J. DUNDAS, M.A., M.D., 8, Upper Wimpole street, Cavendish square.

1868 GREEN, T. HENRY, M.D., Physician to Charing Cross Hospital, and to the Hospital for Consumption, Brompton; 74, Wimpole street, Cavendish square. C. 1886. *Referee*, 1882-5.

1885 GRIFFITH, WALTER SPENCER ANDERSON, M.D., Assistant Physician-Accoucheur, St. Bartholomew's Hospital; University Lecturer and Examiner in Obstetrics, Cambridge; 114, Harley street, Cavendish square.

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1868 GRIGG, WILLIAM CHAPMAN, M.D., Obstetric Physician to the Out-patients at the Westminster Hospital; Physician to the In-Patients, Queen Charlotte's Lying-in Hospital; Joint Lecturer on Forensic Medicine at the Westminster Hospital Medical School; 27, Curzon street, Mayfair.

1889 GUBB, ALFRED S, M.D.Paris; 29, Gower street.

1883 GUNN, ROBERT MARCUS, M.A., M.B., Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Ophthalmic Surgeon to the Hospital for Sick Children, Great Ormond Street, and to the National Hospital for the Paralysed and Epileptic; Assistant Ophthalmic Surgeon to University College Hospital; 54, Queen Anne street, Cavendish square.

1890 GUTHRIE, LEONARD GEORGE, M.B., B.S., Physician to the Regent's Park Hospital for Paralysis; Assistant Physician to the North-West London Hospital; Assistant Physician and Pathologist to the Children's Hospital, Paddington Green; 15, Upper Berkeley street, Portman square.

1886 HABERSHON, SAMUEL HERBERT, M.D., Assistant Physician to the Hospital for Consumption, Brompton; 70, Brook street, Grosvenor square.

1885 HAIG, ALEXANDER, M.D., Physician to the Metropolitan Hospital, and to the Royal Hospital for Children and Women; 7, Brook street, Grosvenor square. *Trans.* 6.

1890 HALE, CHARLES DOUGLAS BOWDICH, M.D., 3, Sussex place, Hyde Park.

1881 HALL, FRANCIS DE HAVILLAND, M.D., Physician to Out-patients and to the Throat Department at the Westminster Hospital; Physician to St. Mark's Hospital; 47, Wimpole street, Cavendish square. *Referee*, 1893—.

1891 HAMER, WILLIAM HEATON, M.D., 73, Dartmouth Park Hill, Highgate.

1889 HANDFIELD-JONES, MONTAGU, M.D., Obstetric Physician to, and Lecturer on Midwifery and Diseases of Women at, St. Mary's Hospital; Physician to the British Lying-in Hospital; 35, Cavendish square.

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†1856 HARE, CHARLES JOHN, M.D., Consulting Physician to University College Hospital; Berkeley House, 15, Manchester square. C. 1873-4. T. 1887-94. V.P. 1894—. *Bldg. Com.* 1889-92.

†1857 HARLEY, GEORGE, M.D., F.R.S. 25, Harley street, Cavendish square. C. 1871-2. V.P. 1891-2. *Referee*, 1865-70, 1873-6. *Sci. Com.* 1862-3, 1866, 1889—. *Trans.* 1.

1864 HARLEY, JOHN, M.D., F.L.S., Physician to, and Lecturer on Clinical Medicine at, St. Thomas's Hospital; 9, Stratford place, Oxford street. S. 1875-7. C. 1879-80. *Referee*, 1871-4, 1882—. *Sci. Com.* 1879. *Trans.* 10.

1893 HARLEY, VAUGHAN, M.D., 25, Harley street, W.

1892 HAROLD, JOHN, 91, Harley street, Cavendish square.

1880 HARRIS, VINCENT DORMER, M.D., Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; Demonstrator of Physiology at St. Bartholomew's Hospital; 31, Wimpole street, Cavendish square.

1870 HARRISON, REGINALD, 6, Lower Berkeley Street, Portman square. C. 1894—. *Trans.* 1.

1870 HAWARD, J. WARRINGTON, *Trustee for Debenture-holders*, Surgeon to, and Lecturer on Clinical Surgery at. St. George's Hospital; 16, Savile row, Burlington Gardens. C. 1885. S. 1888-91. V.P. 1894—. *Lib. Com.* 1881-4. *Sci. Com.* 1889-91. *Bldg. Com.* (*Sec.*) 1889-92. *Ho. Com.* 1892—. *Trans.* 2.

1891 HAWKINS, HERBERT PENNELL, M.B., B.C., Assistant Physician to St. Thomas's Hospital; 38, Weymouth street, Portland place.

1875 HAYES, THOMAS CRAWFORD, M.A., M.D., Physician-Accoucheur and Physician for Diseases of Women and Children to King's College Hospital, and Lecturer in Practical Obstetrics in King's College; Physician for Diseases of Women to Royal Free Hospital; 17, Clarges street, Piccadilly.

1860 HAYWARD, HENRY HOWARD, Consulting Surgeon Dentist to St. Mary's Hospital; 38, Harley street, Cavendish square. C. 1878-9.

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†1865 HEATH, CHRISTOPHER, Holme Professor of Clinical Surgery in University College, London; and Surgeon to University College Hospital; 36, Cavendish square. C. 1880. V.P. 1889. *Lib. Com.* 1870-3. *Trans.* 3.

1882 HENSLEY, PHILIP JOHN., M.D., Assistant Physician and Lecturer on Forensic Medicine to St. Bartholomew's Hospital; 4, Henrietta street, Cavendish square.

1877 HERMAN, GEORGE ERNEST, M.B., Obstetric Physician to and Lecturer on Midwifery at, the London Hospital; 20, Harley street, Cavendish square. *Referee*, 1892—. *Trans.* 1.

1877 HERON, GEORGE ALLAN, M.D., Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; 57, Harley street, Cavendish square.

1891 HERRING, HERBERT T., M.B., B.S., 50, Harley street, Cavendish square.

1883 HERRINGHAM, WILMOT PARKER, M.D., Medical Registrar, St. Bartholomew's Hospital; 13, Upper Wimpole street, Cavendish square. *Trans.* 1.

1893 HERSCHELL, GEORGE, M.D., 25, Queen Anne street, Cavendish square.

1887 HEWITT, FREDERIC WILLIAM, M.D., Instructor in, and Lecturer on Anæsthetics at, the London Hospital; Anæsthetist at the Dental Hospital of London; 10, George street, Hanover square. *Trans.* 1.

1873 HIGGENS, CHARLES, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, Guy's Hospital; 38, Brook street, Grosvenor square. C. 1894—. *Trans.* 2.

1890 HILL, G. WILLIAM, M.D., B.Sc., 24, Wimpole street, Cavendish square.

†1856 HOLMES, TIMOTHY, M.A., Consulting Surgeon to St George's Hospital; Corresponding Member of the Société de Chirurgie, Paris; 18, Great Cumberland place, Hyde Park. C. 1869-70. L. 1873-7. S. 1878-80. V.P. 1881-2. T. 1885-7. P. 1890-92. *Bldg. Com.* (*Chairman*) 1889-92. *Referee*, 1866-8, 1872, 1883-4. *Sci. Com.* 1867. *Lib. Com.* 1863-5, 1892—. *Ho. Com.* 1892—. *Trans.* 8.

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Elected

1878 Hood, Donald William Charles, M.D., Senior Physician to the West London Hospital; 43, Green street, Park lane.

1883 Horsley, Victor Alexander Haden, F.R.S., Surgeon to University College Hospital, Surgeon to the National Hospital for the Paralysed and Epileptic; Professor of Pathology in University College, London; 25, Cavendish square. *Trans.* 1.

1892 Howard, R. J. Bliss, M.D., 31, Queen Anne street, Cavendish square.

1874 Howse, Henry Greenway, M.S., Surgeon to, and Lecturer on Surgery at, Guy's Hospital; Consulting Surgeon to the Evelina Hospital for Sick Children; 59, Brook street, Grosvenor square. C. 1890. *Sci. Com.* 1879. *Referee*, 1887-89. *Trans.* 3.

1886 Hudson, Charles Elliott Leopold Barton, Surgical Registrar, Middlesex Hospital; 16, Harley street, Cavendish square.

†1857 Hulke, John Whitaker, F.R.S., *Hon. Librarian*, Surgeon to the Middlesex Hospital; Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 10, Old Burlington street. C. 1871-2. S. 1876-7. L. 1879—. *Sci. Com.* 1867. *Lib. Com.* 1864-8. *Trans.* 11.

1889 Humphery, Francis William, M.A., M.B., 63, Prince's gate.

1889 Hunter, William, M.D., Assistant Physician to the London Fever Hospital; 54, Harley street.

1873 Hunter, Sir W. Guyer, M.D., K.C.M.G., Hon. Surgeon to H.M. the Queen; formerly Principal of, and Professor of Medicine in, Grant Medical College, and Vice-Chancellor of the University, Bombay; Surgeon-General (Retired) Bombay Army; Consulting Physician to Charing Cross Hospital; 21, Norfolk crescent, Hyde Park.

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†1856 HUTCHINSON, JONATHAN, F.R.S., *President*, Consulting Surgeon to, and Emeritus Professor of Surgery at, the London Hospital; Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; and Surgeon to the Hospital for Diseases of the Skin; 15, Cavendish square. C. 1870. V.P. 1882. P. 1894—. *Referee*, 1876-81, 1883-94. *Lib. Com.* 1864-5. *Trans.* 14. *Pro.* 2.

1888 HUTCHINSON, JONATHAN, Jun., Assistant Surgeon to the London Hospital; 1, Park crescent, W. *Trans.* 1.

1871 JACKSON, J. HUGHLINGS, M.D., F.R.S., Physician to the London Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 3, Manchester square. C. 1889.

†1841 JACKSON, PAUL, 51, Wellington road, St. John's Wood. C. 1862.

1883 JACOBSON, WALTER HAMILTON ACLAND, M.A., M.B., M.Ch., Assistant Surgeon and Lecturer on Anatomy to Guy's Hospital; Surgeon to the Royal Hospital for Children and Women; 66, Great Cumberland place, Hyde Park. *Trans.* 2.

1892 JAMES, EDWIN MATTHEWS, Belgrave Mansions, Grosvenor gardens.

1884 JENNINGS, CHARLES EGERTON, M.D., M.S., 48, Seymour street, Portman square.

1884 JESSETT, FREDERIC BOWREMAN, Surgeon to the Cancer Hospital, Brompton; 1, Buckingham Palace Mansions.

1883 JESSOP, WALTER H. H., M.B., Ophthalmic Surgeon to St. Bartholomew's Hospital; 73, Harley street.

†1847 JOHNSON, SIR GEORGE, M.D., F.R.S., Physician Extraordinary to H.M. the Queen; Consulting Physician to King's College Hospital; Emeritus Professor of Clinical Medicine; Fellow and Member of the Council, King's College, London; Member of the Senate of the University of London; 11, Savile row, Burlington gardens. C. 1862-3. V.P. 1870. P. 1884-5. L. 1878-80. *Referee*, 1853-61, 1864-9. *Lib. Com.* 1860-1. *Trans.* 10. *Pro.* 1.

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Elected

1881 JOHNSON, GEORGE LINDSAY, M.A., M.D., Cortina, Netherhall gardens, South Hampstead, and 14, Stratford place, Oxford street.

1889 JOHNSON, RAYMOND, M.B., B.S., Assistant Surgeon to University College Hospital and Surgeon to the Victoria Hospital for Children; 20, Weymouth street. *Trans.* 1.

1884 JOHNSTON, JAMES, M.D., 11, Chester place, Hyde Park square.

1887 JONES, HENRY LEWIS, M.D., Medical Officer in charge of Electrical Department at St. Bartholomew's Hospital; 9, Upper Wimpole street, Cavendish square.

1881 JULER, HENRY EDWARD, Ophthalmic Surgeon to St. Mary's Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital; Consulting Ophthalmic Surgeon to the London Lock Hospital; 23, Cavendish square.

1893 KANTHACK, ALFRED A., M.D., St. Bartholomew's Hospital, E.C.

1882 KEETLEY, CHARLES R. B., Senior Surgeon to the West London Hospital; 56, Grosvenor street, Grosvenor square.

1884 KESER, JEAN SAMUEL, M.D., Physician to the French Hospital, Shaftesbury avenue, W.C.; 11, Harley street, Cavendish square.

†1857 KIALLMARK, HENRY WALTER, 5, Pembridge gardens. C. 1890-91.

1881 KIDD, PERCY, M.A., M.D., Physician to the Hospital for Consumption, Brompton, and Assistant Physician to the London Hospital; 60, Brook street, Grosvenor square. *Trans.* 4.

†1851 KINGDON, JOHN ABERNETHY, Consulting Surgeon to the Bank of England; 2, Bank buildings, Lothbury. C. 1866-7. V.P. 1872-3. *Sci. Com.* 1867. *Trans.* 1.

1884 LANE, WILLIAM ARBUTHNOT, M.S., Assistant Surgeon to Guy's Hospital and to the Hospital for Sick Children; 8, St. Thomas's street, Southwark. *Trans.* 3.

1882 LANG, WILLIAM, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, the Middlesex Hospital; Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 22, Cavendish square.

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Elected

†1863 LANGDON-DOWN, JOHN LANGDON HAYDON, M.D., Consulting Physician to the London Hospital; 81, Harley street, Cavendish square. C. 1880. V.P. 1890-91. *Trans.* 2.

1865 LANGTON, JOHN, Surgeon to, and Lecturer on Clinical Surgery at, St. Bartholomew's Hospital; Surgeon to the City of London Truss Society; 62, Harley street, Cavendish square. C. 1881-2. *Referee*, 1885—. *Lib. Com.* 1879-80, 1888—. *Trans.* 2.

1890 LAW, EDWARD, M.D., C.M., 35, Harley street, Cavendish square.

1888 LAWRENCE, LAURIE ASHER, 125, Harley street, Cavendish square.

1893 LAWSON, ARNOLD, 12, Harley street.

1884 LAWSON, GEORGE, Surgeon-Oculist in Ordinary to H.M. the Queen; Consulting Surgeon to the Royal London Ophthalmic Hospital, and Surgeon to the Middlesex Hospital; 12, Harley street, Cavendish square.

1892 LEADAM, WILLIAM WARD, M.D., 80, Gloucester terrace, Hyde Park.

†1843 LEE, HENRY, Consulting Surgeon to St. George's Hospital; 9, Savile row, Burlington gardens. C. 1856-7. L. 1863-4. V.P. 1868-9. *Referee*, 1855, 1866-8. *Sci. Com.* 1867. *Trans.* 14. *Pro.* 2.

1886 LEWERS, ARTHUR HAMILTON NICHOLSON, M.D., Obstetric Physician to the London Hospital; 60, Wimpole street, Cavendish square. *Trans.* 1.

1878 LISTER, SIR JOSEPH, Bart., D.C.L., LL.D., F.R.S., Surgeon Extraordinary to H.M. the Queen; Emeritus Professor of Clinical Surgery at King's College, London; and Consulting Surgeon to King's College Hospital; 12, Park crescent, Regent's Park. C. 1892.

1891 LITTLE, ERNEST MUIRHEAD, 40, Seymour street, Portman square.

1889 LITTLE, JOHN FLETCHER, M.B., 32, Harley street, Cavendish square.

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Elected

1881 Lockwood, Charles Barrett, Surgeon to the Great Northern Central Hospital; Assistant Surgeon to, and Demonstrator of Operative Surgery at, St. Bartholomew's Hospital; 19, Upper Berkeley street, Portman square. *Trans.* 3.

1881 Lucas, Richard Clement, B.S., M.B., Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; Surgeon to the Evelina Hospital for Sick Children; Corresponding Member of the Société de Chirurgie of Paris; 18, Finsbury square. *Trans.* 2.

1888 Luff, Arthur Pearson, M.D., B.Sc., Physician to Out-patients and Lecturer on Medical Jurisprudence at St. Mary's Hospital; Official Analyst to the Home Office; 31, Weymouth street, Portland place.

1887 Lush, Percy J. F., M.B., 4, Maresfield gardens, Hampstead.

†1873 MacCarthy, Jeremiah, M.A., Surgeon to the London Hospital and Lecturer on Surgery at the London Hospital Medical College; 15, Finsbury square. C. 1886-7. *Lib. Com.* 1882-5. *Referee*, 1890—.

1867 Mac Cormac, Sir William, M.A., M.Ch., D.Sc., Consulting Surgeon to St. Thomas's Hospital; 13, Harley street. C. 1884-5. *Referee*, 1889—. *Trans.* 1.

1880 McHardy, Malcolm Macdonald, Ophthalmic Surgeon to King's College Hospital, and Professor of Ophthalmic Surgery in King's College, London; Surgeon to the Royal Eye Hospital, Southwark; 5, Savile row.

1873 MacKellar, Alexander Oberlin, M.Ch., Surgeon to St. Thomas's Hospital; Surgeon-in-Chief to the Metropolitan Police Force; 79, Wimpole street, Cavendish square.

1881 Mackenzie, Stephen, M.D., Physician to the London Hospital, and Lecturer on the Principles and Practice of Medicine at the London Hospital Medical College; Physician to the Royal London Ophthalmic Hospital; 18, Cavendish square. *Referee*, 1890—. *Trans.* 1.

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Elected

1879 MACLAGAN, THOMAS JOHN, M.D., Physician-in-Ordinary to their R.H. the Prince and Princess Christian of Schleswig-Holstein ; 9, Cadogan place, Belgrave square.

1889 MACLEHOSE, NORMAN MACMILLAN, M.B., C.M., 13, Queen Anne street, Cavendish square.

1893 MCLEOD, KENNETH, M.D., 39, Clanricarde gardens, Bayswater. *Trans.* 1.

1876 MACNAMARA, CHARLES N., Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital; Surgeon-Major Bengal Medical Service; Fellow of the Calcutta University; 13, Grosvenor street. C. 1891-2. *Referee*, 1884-90. *Lib. Com.* 1886-90.

1881 MACREADY, JONATHAN FORSTER CHRISTIAN HORACE, Surgeon to the Great Northern Hospital; 51, Queen Anne street, Cavendish square.

1880 MADDICK, EDMUND DISTIN, 2, Chandos street, Cavendish square.

1886 MAGUIRE, ROBERT, M.D., Physician to Out-patients and Joint Lecturer on Pathology at St. Mary's Hospital; Assistant Physician to the Hospital for Consumption, Brompton; 4, Seymour street, Portman square. *Sci. Com.* 1889—.

1880 MAKINS, GEORGE HENRY, Assistant Surgeon to St. Thomas's Hospital and Surgeon to the Evelina Hospital for Children ; 47, Charles street, Berkeley square. *Trans.* 1.

1885 MALCOLM, JOHN DAVID, M.B., C.M., Surgeon to the Samaritan Free Hospital; 13, Portman street, Portman square. *Trans.* 1.

1890 MANSON, PATRICK, M.D., C.M., LL.D., Physician to the Seamen's Hospital, Greenwich ; 21, Queen Anne street, Cavendish square.

1855 MARCET, WILLIAM, M.D., F.R.S., Flowermead, Wimbledon Park. C. 1871. *Referee*, 1866-70, 1883-6. *Sci. Com.* 1863. *Lib. Com.* 1866-8. *Trans.* 3.

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1867 MARSH, F. HOWARD, Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital; 30, Bruton street, Berkeley square. C. 1882-3, 1889. S. 1885-7. V.P. 1891-3. *Lib. Com.* 1880-1. *Trans.* 4.

1891 MARTIN, HENRY CHARRINGTON, M.D., 27, Oxford square.

1884 MARTIN, SIDNEY HARRIS COX, M.D., Assistant Physician to University College Hospital, and to the Hospital for Consumption, Brompton; 10, Mansfield street, Portland place.

1892 MASTERS, JOHN ALFRED, M.D., 57, Lexham gardens, Kensington.

1892 MAUNSELL, HENRY WIDENHAM, M.A., M.D.Dublin, 102, Cromwell road, South Kensington, S.W.

1891 MAY, WILLIAM PAGE, M.D., B.Sc., 38, Weymouth street.

1891 MERCIER, CHARLES ARTHUR, M.B., Lecturer on Neurology and Insanity to Westminster Hospital; 8, New Court, Lincoln's Inn, and Flower House, Southend, Catford.

1880 MEREDITH, WILLIAM APPLETON, M.B., C.M., Surgeon to the Samaritan Free Hospital for Women and Children; 21, Manchester square. *Trans.* 1.

1893 MILEY, MILES, M.B., 21, Belsize avenue, Hampstead.

1891 MOLINE, PAUL, M.B., 42, Walton street, Chelsea.

1873 MOORE, NORMAN, M.D., Assistant Physician and Joint Lecturer on Medicine at St. Bartholomew's Hospital; 94, Gloucester place, Portman square. C. 1891-2. *Referee*, 1886-90. *Sci. Com.* 1889—.

1878 MORGAN, JOHN HAMMOND, M.A., Surgeon to the Charing Cross Hospital and to the Hospital for Sick Children, Great Ormond street; 68, Grosvenor street. *Trans.* 2.

1893 MORISON, ALEXANDER, M.D., 14, Upper Berkeley street, W.

1874 MORRIS, HENRY, M.A., Surgeon to, and Lecturer on Surgery at, the Middlesex Hospital; 8, Cavendish square. C. 1888-9. *Referee*, 1882-7. *Trans.* 10.

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1879 MORRIS, MALCOLM ALEXANDER, Surgeon to the Skin Department of, and Lecturer on Dermatology at, St. Mary's Hospital; 8, Harley street, Cavendish square. *Sci. Com.* 1889—. *Trans.* 1.

1885 MOTT, FREDERICK WALKER, M.D., Assistant Physician and Lecturer on Physiology, Charing Cross Hospital; 84, Wimpole street, Cavendish square.

†1888 MURRAY, HUBERT MONTAGUE, M.D., Physician to the Charing Cross Hospital; 27, Savile row.

1880 MURRELL, WILLIAM, M.D., Physician to Out-patients, and Lecturer on Materia Medica and Therapeutics at the Westminster Hospital; 17, Welbeck street, Cavendish square. *Sci. Com.* 1889—. *Trans.* 1.

1892 MYDDELTON-GAVEY, E. HERBERT, 94, Wimpole street, Cavendish square.

1863 MYERS, ARTHUR BOWEN RICHARDS, late Brigade-Surgeon, Brigade of Guards; 43, Gloucester street, Warwick square. C. 1878-9. *Lib. Com.* 1877.

1877 NETTLESHIP, EDWARD, Ophthalmic Surgeon to, and Lecturer on Ophthalmology at, St. Thomas's Hospital; Surgeon to the Royal London Ophthalmic Hospital; 5, Wimpole street, Cavendish square. *Referee*, 1892—.

1864 NUNN, THOMAS WILLIAM, Consulting Surgeon to the Middlesex Hospital; 8, Stratford place, Oxford street.

1870 NUNNELEY, FREDERICK BARHAM, M.D. *Trans.* 2.

1880 OGILVIE, GEORGE, M.B., B.Sc., Physician to the Hospital for Epilepsy and Paralysis, Regent's Park; 22, Welbeck street, Cavendish square.

1880 OGILVIE, LESLIE, M.B., B.Sc., Physician to the Paddington Green Children's Hospital; 46, Welbeck street, Cavendish square.

1891 OGLE, CYRIL, M.A., M.B., 30, Cavendish square.

1858 OGLE, JOHN WILLIAM, M.A., M.D., Consulting Physician to St. George's Hospital; 30, Cavendish square. C. 1873. V.P. 1886. *Referee*, 1864-72. *Trans.* 4.

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1860 OGLE, WILLIAM, M.D., Late Superintendent of Statistics in the Registrar-General's Department, Somerset House; 10, Gordon street, Gordon square. S. 1868-70. C. 1876-7. V.P. 1887. *Lib. Com.* 1871-5. *Trans.* 5.

1892 OPENSHAW, T. HORROCKS, M.B., M.S., Assistant Surgeon to, and Senior Demonstrator of Anatomy at, the London Hospital; 16, Wimpole street, Cavendish square.

1873 ORD, WILLIAM MILLER, M.D., Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; 37, Upper Brook street, Grosvenor square. C. 1889–90. *Sci. Com.* 1889—. *Referee*, 1884-8. *Trans.* 6.

1890 ORD, WILLIAM WALLIS, M.D., 2, Queen street, Mayfair.

1877 ORMEROD, JOSEPH ARDERNE, M.D., Physician to the National Hospital for the Paralysed and Epileptic, Queen square, and to the City of London Hospital for Diseases of the Chest, Victoria Park; 25, Upper Wimpole street. *Trans.* 1.

1875 OSBORN, SAMUEL C., 10, Maddox street, Regent street.

1879 OWEN, EDMUND, M.B., Surgeon to, and Joint Lecturer on Surgery at St. Mary's Hospital; Senior Surgeon to the Hospital for Sick Children, Great Ormond street; 64, Great Cumberland place, Hyde park. *Trans.* 3.

1882 OWEN, HERBERT ISAMBARD, M.D., Assistant Physician to, and Lecturer on Forensic Medicine at, St. George's Hospital; 40, Curzon street, Mayfair. *Bldg. Com.* 1889-92. *Referee* 1893.

1892 PAGE, HARRY MARMADUKE, 82, Ashley gardens, Victoria street.

1874 PAGE, HERBERT WILLIAM, M.A., M.C., Surgeon to, and Joint Lecturer on Surgery at, St. Mary's Hospital; 146, Harley street, Cavendish square. C. 1890-91. *Referee*, 1884-89. *Lib. Com.* 1886-8. *Trans.* 4.

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Elected

†1840 PAGET, SIR JAMES, Bart., D.C.L., LL.D., F.R.S., Sergeant-Surgeon to H.M. the Queen; Surgeon-in-Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. Bartholomew's Hospital; Vice-Chancellor of the University of London; Foreign Associate of the Académie de Médecine, Paris; 5, Park place, Regent's park. C. 1848-9. V.P. 1861. T. 1867. P. 1875-6. *Referee*, 1844-6, 1848, 1851-60, 1862-6, 1868-74. *Sci. Com.* 1863. *Lib. Com.* 1846-7. *Trans.* 12.

1886 PAGET, STEPHEN, Surgeon to the West London Hospital; 57, Wimpole street, Cavendish square.

1873 PARKER, ROBERT WILLIAM, Senior Surgeon to the East London Hospital for Children; Surgeon to the German Hospital; 13, Welbeck street, Cavendish square. C. 1888-9. *Bldg. Com.* 1889-92. *Referee*, 1891—. *Lib. Com.* 1885-87, 1892—. *Ho. Com.* 1892—. *Trans.* 4.

1889 PARSONS, J. INGLIS, M.D., Physician to Out-patients, Chelsea Hospital for Women; 3, Queen street, Mayfair.

1883 PASTEUR, WILLIAM, M.D., Assistant Physician to the Middlesex Hospital; Physician to the North-Eastern Hospital for Children; 4, Chandos street, Cavendish square.

1891 PATERSON, WILLIAM BROMFIELD, 64, Brook street, Grosvenor square.

1891 PATON, EDWARD PERCY, M.D., St. Bartholomew's Hospital.

1865 PAVY, FREDERICK WILLIAM, M.D., LL.D., F.R.S., Consulting Physician to Guy's Hospital; 35, Grosvenor street. C. 1883-4. V.P. 1893-4. *Referee*, 1871-82. *Trans.* 1.

1869 PAYNE, JOSEPH FRANK, M.D., Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; 78, Wimpole street, Cavendish square. C. 1887. *Referee*, 1890—. *Sci. Com.* 1879. *Lib. Com.* 1878-85, 1889—.

1893 PEGLER, L. HEMINGTON, M.D., 12, Radnor place, Gloucester square, W.

1887 PENROSE, FRANCIS GEORGE, M.D., Assistant Physician to St. George's Hospital; 4, Harley street, Cavendish square. *Sci. Com.* 1889—.

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1890 PERRY, EDWIN COOPER, M.D., Assistant Physician and Demonstrator of Pathology at Guy's Hospital; Superintendent, The College, Guy's Hospital.

1883 PHILLIPS, CHARLES DOUGLAS F., M.D., LL.D., 10, Henrietta street, Cavendish square.

1884 PHILLIPS, GEORGE RICHARD TURNER, 24, Palace Court, Notting hill gate.

1888 PHILLIPS, JOHN, M.A., M.D., Assistant Obstetric Physician, King's College Hospital; Physician to the British Lying-in Hospital; 71, Grosvenor street, Grosvenor square. *Trans.* 1.

1889 PHILLIPS, SIDNEY, M.D., Senior Physician to Out-patients and Lecturer on Materia Medica at St. Mary's Hospital, Senior Physician to the London Fever Hospital, and to the Lock Hospital; 62, Upper Berkeley street, Portman square.

1867 PICK, THOMAS PICKERING, Surgeon to, and Lecturer on Surgery at, St. George's Hospital; 18, Portman street, Portman square. C. 1884-5. V.P. 1893-4. *Referee*, 1882-3, 1891-93. *Sci. Com.* 1870, 1889—. *Lib. Com.* 1879-81.

1884 PITT, GEORGE NEWTON, M.D., Assistant Physician to, and Pathologist at, Guy's Hospital; 24, St. Thomas's street, Southwark. *Trans.* 1.

1889 PITTS, BERNARD, M.B., M.C., Surgeon to St. Thomas's Hospital; 109, Harley street, Cavendish square.

1885 POLAND, JOHN, Surgeon to the Miller Hospital, Greenwich; 4, St. Thomas's street, Southwark.

1884 POLLARD, BILTON, B.S., Surgeon to University College Hospital, Surgeon to the North-Eastern Hospital for Children; 24, Harley street, Cavendish square. *Trans.* 1.

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†1845 POLLOCK, GEORGE DAVID, Surgeon-in-Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. George's Hospital; 36, Grosvenor street. C. 1856-7. L. 1859-62. V.P. 1870-1. P. 1886-7. *Referee*, 1858, 1864-9, 1877-85. *Trans.* 5.

1865 POLLOCK, JAMES EDWARD, M.D., Consulting Physician to the Hospital for Consumption, Brompton; 52, Upper Brook street, Grosvenor square. C. 1882-3. *Referee*, 1872-81.

1871 POORE, GEORGE VIVIAN, M.D., Professor of Medical Jurisprudence in University College, London; Physician to University College Hospital; Consulting Physician to the Royal Infirmary for Children and Women, Waterloo road; 30, Wimpole street, Cavendish square. C. 1890-91. *Referee* 1887-89, 1892—. *Trans.* 2.

1885 PORT, HEINRICH, M.D., Physician to the German Hospital; 48, Finsbury square.

1867 POWELL, RICHARD DOUGLAS, M.D., Physician Extraordinary to H.M. the Queen; Physician to the Middlesex Hospital; Consulting Physician to the Hospital for Consumption, Brompton; 62, Wimpole street, Cavendish square. S. (Oct.) 1883-5. C. 1887-8. *Referee* 1879-83, 1886. *Trans.* 3.

1887 POWER, D'ARCY, M.A., M.B., Demonstrator of Surgery at St. Bartholomew's Hospital; Surgeon to the Victoria Hospital for Children; 26, Bloomsbury square. *Trans.* 2.

1867 POWER, HENRY, Senior Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. Bartholomew's Hospital; 37A, Great Cumberland place, Hyde Park. C. 1882-3. V.P. 1892-3. *Referee*, 1870-81, 1891—. *Sci. Com.* 1870, 1889—. *Lib. Com.* 1872-8.

†1857 PRIESTLEY, SIR WILLIAM OVEREND, M.D., LL.D., Consulting Physician to King's College Hospital, and to the West London Hospital and the British Lying-in Hospital; 17, Hertford street, Mayfair. C. 1874-5. V.P. 1884-5. *Referee*, 1867-73, 1877-83. *Sci. Com.* 1863.

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1883 PRINGLE, JOHN JAMES, M.B., C.M., Assistant Physician to, Lecturer on Practical Medicine, and Physician in Charge of Skin Department at, the Middlesex Hospital; 23, Lower Seymour street, Portman square. *Trans.* 1.

1874 PURVES, WILLIAM LAIDLAW, Aural Surgeon to Guy's Hospital; 20, Stratford place, Oxford street. *Trans.* 2.

1877 PYE-SMITH, PHILIP HENRY, M.D., F.R.S., Physician to, and Lecturer on Medicine at, Guy's Hospital; Member of the Senate of the University of London; 48, Brook street, Grosvenor square. C. 1893-94. *Lib. Com.* 1887-93. *Trans.* 1.

†1850 QUAIN, SIR RICHARD, Bart., M.D., (Hon.) M.D.Dublin, LL.D.Ed., F.R.S., Physician Extraordinary to H.M. the Queen; Consulting Physician to the Hospital for Consumption, Brompton, and to the Seamen's Hospital, Greenwich; Member of the Senate of the University of London; 67, Harley street, Cavendish square. C. 1866-7. V.P. 1878-9. *Sci. Com.* 1863. *Trans.* 1.

1871 RALFE, CHARLES HENRY, M.D., M.A., Physician to the London Hospital, and late Physician to the Seamen's Hospital, Greenwich; 26, Queen Anne street, Cavendish square. C. 1889. *Referee*, 1885-8.

1892 RAYNER, HENRY, M.D., 2, Harley street, Cavendish square.

1891 READ, HENRY GEORGE, 30, Finsbury square, and Martins, Shipbourne, Kent.

1869 READ, THOMAS LAURENCE, 11, Petersham terrace, Queen's gate.

1891 REECE, RICHARD JAMES, 31, Holland Villas road, W.

1891 REMFRY, LEONARD, M.A., M.D., Obstetrical Physician to the Great Northern Central Hospital, Assistant Obstetric Physician, and Assistant Lecturer on Obstetric Medicine, St. George's Hospital; 60, Great Cumberland place.

1891 RENDEL, ARTHUR BOWEN, M.A., M.B., B.C., 43, Albion street, Hyde Park, W.

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†1855 REYNOLDS, JOHN RUSSELL, M.D., F.R.S., Physician-in Ordinary to H.M.'s Household; Emeritus Professor of Medicine in University College; Consulting Physician to University College Hospital; 38, Grosvenor street. C. 1870. V.P. 1883. *Referee*, 1867-9.

1887 RICHARDSON, GILBERT, M.A., M.D., Hawthorn House, Putney.

1863 RINGER, SYDNEY, M.D., F.R.S., Holme Professor of Clinical Medicine in University College, London, and Physician to University College Hospital; 15, Cavendish place, Cavendish square. C. 1881-2. *Referee*, 1873-80, 1889—. *Trans.* 6.

1871 RIVINGTON, WALTER, M.S., Consulting Surgeon to the London Hospital; 95, Wimpole street, Cavendish square. C. 1885-6. *Trans.* 5.

1893 ROBERTS, D. WATKIN, M.D., 56, Manchester street, Manchester square.

1878 ROBERTS, FREDERICK THOMAS, M.D., Professor of Materia Medica and Therapeutics, and of Clinical Medicine, in University College, London; and Physician to University College Hospital; Consulting Physician to the Hospital for Consumption, Brompton; 102, Harley street, Cavendish square. C. 1894—. *Sci. Com.* 1889—.

1889 ROBERTS, SIR WILLIAM, M.D., B.A., F.R.S., 8, Manchester square. *Trans.* 2.

1890 ROLLESTON, HUMPHRY DAVY, M.A., M.D., Assistant Physician and Lecturer on Pathology at St. George's Hospital; 13, Upper Wimpole street, Cavendish square.

†1857 ROSE, HENRY COOPER, M.D., Consulting Surgeon to the Hampstead Dispensary; 53, Rosslyn hill, Hampstead. C. 1886-7. *Trans.* 1.

1883 ROSE, WILLIAM, M.B., Professor of Clinical Surgery in King's College; Surgeon to King's College Hospital; and Consulting Surgeon to the Royal Free Hospital; 17, Harley street, Cavendish square.

1889 ROSS, DANIEL MCCLURE, 24, Cambridge street, Hyde Park.

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1888 ROUGHTON, EDMUND WILKINSON, B.S., M.D., Warden of the College of St. Mary's Hospital; Assistant Surgeon to the Royal Free Hospital; 33, Westbourne terrace, Hyde Park. *Trans.* 1.

1882 ROUTH, AMAND JULES MCCONNEL, M.D., B.S., Physician to the Samaritan Free Hospital for Women; Obstetric Physician to Out-patients, and Lecturer on Practical Midwifery at the Charing Cross Hospital; 14A, Manchester square.

†1849 ROUTH, CHARLES HENRY FELIX, M.D., Consulting Physician to the Samaritan Free Hospital for Women and Children; 52, Montagu square. *Lib. Com.* 1854-5. *Trans.* 1.

1891 RUFFER, MARC ARMAND, M.A., M.D., 5, York terrace, Regent's park.

1891 RUSSELL, J. S. RISIEN, M.B., C.M., 4, Queen Anne street, Cavendish square.

1886 SAINSBURY, HARRINGTON, M.D., Physician to the Royal Free Hospital and Assistant Physician to the City of London Hospital for Diseases of the Chest; 63, Welbeck street, Cavendish square. *Trans.* 1.

1869 SANSOM, ARTHUR ERNEST, M.D., Physician to the London Hospital; Consulting Physician and Vice-President, North-Eastern Hospital for Children; 84, Harley street, Cavendish sq. C. 1887-8. *Referee*, 1889—. *Trans.* 3.

1893 DE SANTI, PHILIP ROBERT WILLIAM, 37, Queen Anne street, Cavendish square.

†1845 SAUNDERS, SIR EDWIN, Surgeon-Dentist to H.M. the Queen, and to their R.H. the Prince and Princess of Wales; Fairlawn, Wimbledon Common. C. 1872-3.

1879 SAVAGE, GEORGE HENRY, M.D., Lecturer on Mental Diseases at Guy's Hospital; 3, Henrietta street, Cavendish square.

1859 SAVORY, SIR WILLIAM, Bart., F.R.S., Surgeon Extraordinary to H.M. the Queen; Consulting Surgeon to St. Bartholomew's Hospital; 66, Brook street, Grosvenor square. C. 1871-2. L. 1878. V.P. 1883-4. *Referee*, 1865-70, 1873-77, 1879-82. *Sci. Com.* 1862, 1867, 1870. *Lib. Com.* 1866-8. *Trans.* 8.

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1883 SCHÄFER, EDWARD ALBERT, F.R.S., Jodrell Professor of Physiology, University College, London; University College, Gower street. *Referee*, 1888—. *Sci. Com.* 1889—.

1892 SCHORSTEIN, GUSTAVE, M.A., M.B., B.Ch., D.Ph., Assistant Physician to the London Hospital and to the Hospital for Consumption, Brompton; 11, Portland place.

1887 SCOTT, HARRY, M.D., 27, St. Ermin's mansions, Westminster.

1882 SCRIVEN, JOHN BARCLAY, Brigade Surgeon, Bengal (retired), late Professor of Anatomy, Surgery, and Ophthalmic Surgery at the Lahore Medical School; 95, Oxford gardens, Notting hill.

1863 SEDGWICK, WILLIAM, 101, Gloucester place, Portman square. C. 1884-5. *Trans.* 3.

1892 DE SEGUNDO, CHARLES SEMPILL, 2, Aldridge road villas, Westbourne park.

1892 SELWYN-HARVEY, JOHN STEPHENSON, M.D., 1, Astwood road, Cromwell road.

1877 SEMON, FELIX, M.D., Physician for Diseases of the Throat to St. Thomas's Hospital; 39, Wimpole street, Cavendish square. *Lib. Com.* 1894—. *Trans.* 2.

1893 SEWILL, JOSEPH SEFTON, 9A, Cavendish square.

1882 SHARKEY, SEYMOUR JOHN, M.D., Physician and Joint Lecturer on Pathology at St. Thomas's Hospital; 2, Portland place. *Trans.* 2.

1886 SHAW, LAURISTON ELGIE, M.D., Assistant Physician to Guy's Hospital; 10, St. Thomas's street, Southwark.

1884 SHEILD, ARTHUR MARMADUKE, M.B., B.S., Assistant Surgeon, St. George's Hospital; 4, Cavendish place. *Trans.* 3.

1893 SIBLEY, WALTER KNOWSLEY, M.D., 7, Upper Brook street.

†1848 SIEVEKING, SIR EDWARD HENRY, M.D., LL.D., F.S.A., Physician-in-Ordinary to H.M. the Queen; Physician-in-Ordinary to H.R.H. the Prince of Wales; Consulting Physician to St. Mary's and the Lock Hospitals; 17, Manchester square. C. 1859-60. S. 1861-3. V.P. 1873-4. L. 1881-2. P. 1888-9. *Referee*, 1855-8, 1864-72, 1875-80. *Sci. Com.* 1862. *Trans.* 2.

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1886 SILCOCK, ARTHUR QUARRY, M.D., B.S., Surgeon in charge of Out-patients, St. Mary's Hospital; Surgeon, Royal London Ophthalmic Hospital; 52, Harley street, Cavendish square.

†1842 SIMON, SIR JOHN, K.C.B., D.C.L., LL.D., F.R.S., Consulting Surgeon to St. Thomas's Hospital; 40, Kensington square. C. 1854-5. V.P. 1865. *Referee* 1851-3, 1866-81. *Trans.* 1.

1892 SIMS, FRANCIS MANLEY BOLDERO, 12, Hertford street, Mayfair.

1893 SISLEY, RICHARD, M.D., 11, York street, Portman square, W.

1890 SMALE, MORTON, 22A, Cavendish square.

1879 SMITH, E. NOBLE, Surgeon to All Saints' Children's Hospital; Orthopædic Surgeon to the British Home for Incurables; 24, Queen Anne street, Cavendish square.

1881 SMITH, EUSTACE, M.D., Physician to H.M. the King of the Belgians; Physician to the East London Children's Hospital, and to the Victoria Park Hospital for Diseases of the Chest; 15, Queen Anne street, Cavendish square.

1891 SMITH, G. COCKBURN, M.D., 5, Inverness gardens, Kensington.

†1838 SMITH, HENRY SPENCER, Consulting Surgeon to St. Mary's Hospital; 92, Oxford terrace, Hyde Park. C. 1854. S. 1855-8. V.P. 1859-60. T. 1865. *Referee*, 1851-3, 1862-4, 1866-78. *Lib. Com.* 1847.

1866 SMITH, HEYWOOD, M.A., M.D., 18, Harley street, Cavendish square.

1889 SMITH, ROBERT PERCY, M.D., B.S., Resident Physician and Medical Superintendent, Bethlem Royal Hospital, St. George's road, Southwark.

1892 SMITH, SOLOMON CHARLES, M.D., 4, Portman Mansions, Baker street.

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1863 SMITH, THOMAS, Surgeon to, and Lecturer on Clinical Surgery at, St. Bartholomew's Hospital; 5, Stratford place, Oxford street. S. 1870-2. C. 1875-6. V.P. 1887-8. *Referee*, 1873-4, 1880-6. *Sci. Com.* 1867. *Trans.* 4.

1872 SMITH, THOMAS GILBART, M.A., M.D., Physician to the London Hospital; Physician to the Royal Hospital for Diseases of the Chest, City road; 68, Harley street, Cavendish square. C. 1890. *Trans.* 1.

1873 SMITH, W. JOHNSON, Surgeon to the Seamen's Hospital Society, Greenwich.

*1874 SMITH, WILLIAM ROBERT, M.D., D.Sc., Barrister-at-Law, Professor of Forensic Medicine in, and Director of the Laboratories of State Medicine at, King's College, London; Medical Officer to the School Board for London; 74, Great Russell Street. *Trans.* 1.

1865 SOUTHEY, REGINALD, M.D., Commissioner in Lunacy; 32, Grosvenor road, Westminster. C. 1881-2. S. 1883. *Referee*, 1873-80. *Trans.* 1.

1889 SPENCER, HERBERT R., M.D., B.S., Professor of Midwifery in University College; Obstetric Physician to University College Hospital; 10, Mansfield street, Portland place. *Referee*, 1894—.

1887 SPENCER, WALTER GEORGE, M.B., M.S., Assistant Surgeon to the Westminster Hospital; 35, Brook street, Grosvenor square. *Trans.* 2.

1888 SPICER, ROBERT HENRY SCANES, M.D., Physician to the Department for Diseases of the Throat, St. Mary's Hospital; 28, Welbeck street, Cavendish square.

1890 SPICER, WILLIAM THOMAS HOLMES, M.B., 47, Welbeck street, Cavendish square.

1875 SPITTA, EDMUND JOHNSON, Ivy House, Clapham Common, Surrey.

†1851 SPITTA, ROBERT JOHN, M.D., 2, The Sweep, East Side, Clapham Common, Surrey. C. 1878-9. *Trans.* 1.

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1885 SQUIRE, JOHN EDWARD, M.D., Physician to the North London Hospital for Consumption; 122, Harley street, Cavendish square. *Trans.* 1.

†1859 STEWART, WILLIAM EDWARD, 16, Harley street, Cavendish square.

†1856 STOCKER, ALONZO HENRY, M.D., Peckham House, Peckham.

1884 STONHAM, CHARLES, Assistant Surgeon to the Westminster Hospital, and Curator of Anatomical Museum; 4, Harley street, Cavendish square.

†1871 SUTHERLAND, HENRY, M.D., Physician to Newland's House and Otto House Private Asylums; 6, Richmond terrace, Whitehall.

1883 SUTTON, JOHN BLAND, Assistant Surgeon to the Middlesex Hospital; 48, Queen Anne street, Cavendish square. *Trans.* 6.

1890 SYERS, HENRY WALTER, M.D., 3, Devonshire street, Portland place.

1886 SYMONDS, CHARTERS JAMES, M.S., Assistant Surgeon to, and Demonstrator of Operative and Practical Surgery at, Guy's Hospital; 26, Weymouth street, Portland place.

1875 TAY, WABEN, Senior Surgeon to the London Hospital; Surgeon to the Royal London Ophthalmic Hospital; Consulting Surgeon to the North-Eastern Hospital for Children, and to the Hospital for Diseases of the Skin, Blackfriars; 4, Finsbury square.

1873 TAYLOR, FREDERICK, M.D., *Trustee;* Physician to, and Lecturer on Medicine at, Guy's Hospital; Consulting Physician to the Evelina Hospital for Sick Children; 20, Wimpole street, Cavendish square. S. 1889-93. C. 1894—. *Sci. Com.* 1889—. *Referee,* 1887-8. *Trans.* 2.

1893 TAYLOR, JAMES, M.D., Assistant Physician to the National Hospital for the Paralysed and Epileptic; 34, Welbeck street, Cavendish square.

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1890 TAYLOR, SEYMOUR, M.D., Assistant Physician West London Hospital; 16, Seymour street, Portman square.

1859 TEGART, EDWARD, 60, Scarsdale Villas, Kensington. C. 1888-9.

1874 THIN, GEORGE, M.D., 22, Queen Anne street, Cavendish square. C. 1893-4. *Trans.* 11.

1862 THOMPSON, EDMUND SYMES, M.D., Consulting Physician to the Hospital for Consumption, Brompton; Gresham Professor of Medicine; 33, Cavendish square. S. 1871-4. C. 1878-9. *Sci. Com.* 1889—. *Referee*, 1876-7. *Trans.* 1.

†1852 THOMPSON, SIR HENRY, Surgeon-Extraordinary to H.M. the King of the Belgians; Emeritus Professor of Clinical Surgery in University College, London; and Consulting Surgeon to University College Hospital; Member of the Société de Chirurgie, Paris; 35, Wimpole street, Cavendish square. C. 1869. *Trans.* 8.

†1862 THOMPSON, REGINALD EDWARD, M.D., Consulting Physician to the Hospital for Consumption, Brompton; 47, Park street, Grosvenor square. C. 1879. S. 1880-82. V.P. 1883-4. *Referee*, 1873-8. *Sci. Com.* 1867. *Trans.* 2.

1892 THOMSON, ST. CLAIR, M.D., 28, Queen Anne street, Cavendish square.

1881 THOMSON, WILLIAM SINCLAIR, M.D. (Travelling).

1892 THORNE, WILLIAM BEZLY, M.D., 5, Gledhow gardens, South Kensington.

1876 THORNTON, JOHN KNOWSLEY, M.B., C.M., Consulting Surgeon to the Samaritan Free Hospital for Women and Children; Consulting Surgeon to the Grosvenor Hospital for Women, and to the New Hospital for Women; 49, Montagu square. C. 1891. *Lib. Com.* 1886-90, 1893—. *Trans.* 5.

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1889 TIRARD, NESTOR ISIDORE CHARLES, M.D., Professor of Materia Medica and Therapeutics, King's College; Physician to King's College Hospital, and Physician to the Evelina Hospital for Sick Children; 74, Harley street, Cavendish square.

1872 TOMES, CHARLES SISSMORE, M.A., F.R.S., 37, Cavendish square. C. 1887. *Lib. Com.* 1879.

1882 TOOTH, HOWARD HENRY, M.D., Demonstrator of Morbid Anatomy, St. Bartholomew's Hospital; Physician to the Metropolitan Hospital; Assistant Physician to the National Hospital for the Paralysed and Epileptic, Queen square; 34, Harley street, Cavendish square.

1879 TREVES, FREDERICK, Surgeon to, and Lecturer on Anatomy at, the London Hospital; 6, Wimpole street, Cavendish square. *Referee*, 1890—. *Sci. Com.* 1889—. *Trans.* 5.

1859 TRUMAN, EDWIN THOMAS, Surgeon-Dentist in Ordinary to Her Majesty's Household; 23, Old Burlington street.

1889 TURNBULL, GEORGE LINDSAY, M.B., Grove House, 76, Ladbroke grove.

1875 TURNER, FRANCIS CHARLEWOOD, M.A., M.D., Physician to the London Hospital, and to the North-Eastern Hospital for Children; 15, Finsbury square.

1882 TURNER, GEORGE ROBERTSON, Visiting Surgeon to the Seamen's Hospital, Greenwich; Assistant Surgeon to, and Lecturer on Anatomy and Joint Lecturer on Practical Surgery at, St. George's Hospital; 49, Green street, Park lane.

1893 TURNER, PHILIP DYMOCK, M.D., 95, Cromwell Road.

1891 TWEED, REGINALD, M.D., 55, Upper Brook street, Grosvenor square.

1892 TWEEDY, JOHN, Professor of Ophthalmic Medicine and Surgery in University College, Ophthalmic Surgeon to University College Hospital, and to the Royal London Ophthalmic Hospital; 100, Harley Street, Cavendish square.

1876 VENN, ALBERT JOHN, M.D., Physician for the Diseases of Women, West London Hospital; 122, Harley street, Cavendish square.

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1870 VENNING, EDGCOMBE, 30, Cadogan place.

1865 VERNON, BOWATER JOHN, Ophthalmic Surgeon to St. Bartholomew's Hospital and to the West London Hospital; 14, Clarges street, Piccadilly.

1867 VINTRAS, ACHILLE, M.D., Physician to the French Embassy, and Senior Physician to the French Hospital and Dispensary, Shaftesbury Avenue; 19A, Hanover square.

1891 VOELCKER, ARTHUR FRANCIS, M.D., B.S., Pathologist and Curator of the Museum at the Middlesex Hospital; 31, Harley street, W.

1886 WAINEWRIGHT, BENJAMIN, M.B., C.M., Assistant Surgeon to the Royal Westminster Ophthalmic Hospital; 67, Grosvenor street, Grosvenor square.

1884 WAKLEY, THOMAS, jun., 5, Queen's Gate, South Kensington.

1883 WALLER, AUGUSTUS, M.D., F.R.S., Lecturer on Physiology, St. Mary's Hospital; Weston Lodge, 16, Grove End road.

1888 WALLIS, FREDERICK CHARLES, M.B., B.C., Assistant Surgeon to Charing Cross Hospital; 26, Welbeck street, Cavendish square.

1873 WALSHAM, WILLIAM JOHNSON, C.M., Senior Assistant Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; Surgeon to the Metropolitan Free Hospital; 77, Harley street, Cavendish square. C. 1888-9. *Lib. Com.* 1882-5. *Trans.* 6.

1886 WARD, ALLAN OGIER, M.D., Lansdowne House, High road, Tottenham.

1890 WARD, ARTHUR HENRY, Surgeon to Out-patients, Lock Hospital; 7, Hertford street, Mayfair.

1891 WARING, HOLBURT JACOB, M.B., B.S., B.Sc., 9, Upper Wimpole street.

1877 WARNER, FRANCIS, M.D., Physician and Lecturer on Materia Medica and Therapeutics to the London Hospital; 5, Prince of Wales terrace, Kensington Palace, *Trans.* 1.

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1889 WASHBOURN, JOHN WYCHENFORD, M.D., Assistant Physician to, Physician in Charge of Electrical Department, Joint Lecturer on Physiology, and Demonstrator of Bacteriology at Guy's Hospital; Physician to the London Fever Hospital; 15, Trinity square, S.E. *Trans.* 1.

1893 WATERHOUSE HERBERT FURNIVALL, M.D., Assistant Surgeon to Charing Cross Hospital; 81, Wimpole street, W.

†1861 WATSON, WILLIAM SPENCER, M.B., Surgeon to the Throat Department of the Great Northern Central Hospital; Surgeon to the Royal Eye Hospital; 7, Henrietta street, Cavendish square. C. 1883-4. *Trans.* 1.

1879 DE WATTEVILLE, ARMAND, M.A., M.D., B.Sc., 30, Welbeck street, Cavendish square.

1892 WEAVER, FREDERICK POYNTON, M.D., Cedar Lawn, Hampstead Heath.

†1891 WEBER, FREDERIC PARKES, M.D., 19, Harley street, W.

1857 WEBER, HERMANN, M.D., Consulting Physician to the German Hospital; 10, Grosvenor street, Grosvenor square. C. 1874-5. V.P. 1885-6. *Sci. Com.* 1889—. *Referee*, 1869-73, 1878-84. *Lib. Com.* 1864-73 *Trans.* 6.

†1854 WELLS, SIR THOMAS SPENCER, Bart., Hon. M.D. Dublin, Leyden, Bologna, Charkoff; Surgeon-in-Ordinary to H.M.'s Household; Consulting Surgeon to the Samaritan Free Hospital for Women and Children; Foreign Associate, Académie de Médecine, Paris; 3, Upper Grosvenor street. C. 1870. V.P. 1881. *Trans.* 14. *Pro.* 1.

†1842 WEST, CHARLES, M.D., Foreign Associate, Académie de Médecine, Paris; 4, Evelyn Mansions, Carlyle place, Victoria street, S.W. C. 1855-6. V.P. 1863. P. 1877-8. *Referee*, 1848-54, 1857-62, 1864-76, 1880. *Sci. Com.* 1863. *Lib. Com.* 1844-7, 1851. *Trans.* 2.

1877 WEST, SAMUEL, M.D., Assistant Physician to St. Bartholomew's Hospital; Senior Physician to the Royal Free Hospital; 15, Wimpole street, Cavendish square. C. 1894—. *Lib. Com.* 1892—4. *Trans.* 4.

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1888 WETHERED, FRANK JOSEPH, M.D., Assistant Physician to the Hospital for Consumption, Brompton; 34, Queen Anne street, Cavendish square. *Trans.* 1.

1881 WHARRY, ROBERT, M.D., 6, Gordon square.

1878 WHARTON, HENRY THORNTON, M.A., Senior Honorary Surgeon to the Kilburn Dispensary; "Madresfield," Acol road, Priory road, West Hampstead.

1875 WHIPHAM, THOMAS TILLYER, M.D., Physician to, and Lecturer on Medicine at, St. George's Hospital; 11, Grosvenor street, Grosvenor square. C. 1892-3.

1891 WHITE, CHARLES PERCIVAL, M.B., B.C., 144, Sloane street.

1881 WHITE, WILLIAM HALE, M.D., Physician to, and Lecturer on Materia Medica at, Guy's Hospital; 65, Harley street, Cavendish square. *Referee*, 1888—. *Trans.* 4.

1890 WHITE-COOPER, G. O., M.B., 5, Courtfield road, Gloucester road, S.W.

1877 WHITMORE, WILLIAM TICKLE, Senior Surgeon to the Westminster General Dispensary, to the St. George's and St. James's Dispensary, and to the Gordon Hospital for Diseases of the Rectum; 7, Arlington street, Piccadilly.

1863 WILKS, SAMUEL, M.D., LL.D., F.R.S., Physician in Ordinary to their Royal Highnesses the Duke and Duchess of Connaught; Consulting Physician to Guy's Hospital, and Member of the Senate of the University of London; 72, Grosvenor street, Grosvenor square. *Referee*, 1872–81. *Sci. Com.* 1.

1890 WILLCOCKS, FREDERICK, M.D., Physician to Out-Patients, and Lecturer on Materia Medica and Therapeutics, at the Charing Cross Hospital; Physician to the Evelina Hospital for Sick Children; 14, Mandeville place, Manchester square.

†1865 WILLETT, ALFRED, *Trustee*, Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital; Surgeon to St. Luke's Hospital; 36, Wimpole street, Cavendish square. C. 1880-81. V.P. 1890-91. *Referee*, 1882-89, 1892—. *Bldg. Com.* 1889-92. *Ho. Com.* 1892—. *Trans.* 2

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1887 WILLETT, EDGAR, M.B., 25, Welbeck street, Cavendish square.

1888 WILLIAMS, CAMPBELL, 24, Welbeck street, Cavendish square.

1866 WILLIAMS, CHARLES THEODORE, M.A., M.D., *Trustee for Debenture-holders*, Consulting Physician to the Hospital for Consumption and Diseases of the Chest, Brompton; 2, Upper Brook street, Grosvenor square. C. 1884-5. *Referee*, 1888—. *Lib. Com.* 1880-3. *Sci Com* 1889—. *Trans.* 5.

1881 WILLIAMS, DAWSON, M.D., Assistant Physician to the East London Hospital for Children; 25, Old Burlington street. *Trans.* 1.

1872 WILLIAMS, SIR JOHN, Bart., M.D., Physician-Accoucheur to H.R.H. the Princess Beatrice; Emeritus Professor of Obstetric Medicine, University College, London; Consulting Obstetric Physician to University College Hospital; 63, Brook street, Grosvenor square. C. 1891. *Referee*, 1878-90. *Lib. Com.* 1876-82.

1890 WILLS, WILLIAM ALFRED, M.D., 23, Lower Seymour street, Portman square.

1879 WOAKES, EDWARD, M.D., Senior Aural Surgeon to the London Hospital; 78, Harley street, Cavendish square.

1885 WOLFENDEN, RICHARD NORRIS, M.D., Physician to the Hospital for Diseases of the Throat, Golden square; 35, Harley street, Cavendish square.

1887 WOOD, THOMAS OUTTERSON, M.D., 40, Margaret street, Cavendish square.

1891 WOODFORDE, ALFRED POWNALL, 160, Goldhawk road.

1892 WOODHEAD, GERMAN SIMS, M.D., 1, Nightingale lane, Balham.

1890 WYNTER, WALTER ESSEX, M.D., B.S., Assistant Physician to the Middlesex Hospital; 30, Upper Berkeley street, Portman square.

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LIST OF RESIDENT FELLOWS

ARRANGED ACCORDING TO

DATE OF ELECTION.

1838 Henry Spencer Smith.
1840 Sir James Paget, Bt., F.R.S.
1841 Paul Jackson.
1842 Sir John Simon, K.C.B., F.R.S.
Charles West, M.D.
John Erichsen, F.R.S.
1843 Henry Lee.
1845 George D. Pollock.
Sir Edwin Saunders.
Edward U. Berry.
1846 John A. Bostock.
1847 Sir G. Johnson, M.D., F.R.S.
1848 Sir Edward H. Sieveking, M.D.
John Clarke, M.D.
1849 C. H. F. Routh, M.D.
1850 Sir R. Quain, Bt., M.D., F.R.S.
1851 John Birkett.
John A. Kingdon.
Peter Y. Gowland.
Bernard E. Brodhurst.
Robert J. Spitta, M.D.
1852 William Adams.
Sir Henry Thompson.
1853 Robert Brudenell Carter.
1854 Sir Alfred B. Garrod, M.D., F.R.S.
Sir Thomas Spencer Wells, Bt.
1855 J. Russell Reynolds, M.D., F.R.S.
William Marcet, M.D., F.R.S.
1856 Charles J. Hare, M.D.
William Bird.
Jonathan Hutchinson, F.R.S.
Timothy Holmes.
Alonzo H. Stocker, M.D.
1857 Sir William Overend Priestley, M.D.
George Harley, M.D., F.R.S.
1857 Hermann Weber, M.D.
John Whitaker Hulke, F.R.S.
Henry Cooper Rose, M.D.
Henry Walter Kiallmark.
1858 John William Ogle, M.D.
1859 Wm. Howship Dickinson, M.D.
Sir William S. Savory, Bt., F.R.S.
Edwin Thomas Truman.
Richard Barwell.
Edward Tegart.
William E. Stewart.
1860 William Ogle, M.D.
Thomas Bryant.
John Couper.
Henry Howard Hayward.
1861 William Spencer Watson.
1862 Lionel Smith Beale, M.B., F.R.S.
Edmund Symes Thompson, M.D.
Reginald Edward Thompson, M.D.
George Cowell.
1863 J. L. H. Langdon-Down, M.D.
Samuel Wilks, M.D., F.R.S.
Samuel Fenwick, M.D.
Julius Althaus, M.D.
Sydney Ringer, M.D., F.R.S.
Thomas Smith.
Arthur B. R. Myers.
Arthur E. Durham.
William Sedgwick.
1864 Sir George Buchanan, M.D., F.R.S.
John Harley, M.D.
Thomas William Nunn.
1865 James Edward Pollock, M.D.
William Cholmeley, M.D.
Reginald Southey, M.D.

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1865 George Fielding Blandford, M.D.
Sir Dyce Duckworth, M.D.
Frederick W. Pavy, M.D., F.R.S.
William Morrant Baker.
John Langton.
Frederick James Gant.
Alfred Willett.
Bowater John Vernon.
Alfred Cooper.
Christopher Heath.
1866 Thomas Fitz-Patrick, M.D.
Samuel Jones Gee, M.D.
Charles Theodore Williams, M.D.
Heywood Smith, M.D.
William Selby Church, M.D.
1867 William Henry Day, M.D.
Achille Vintras, M.D.
Richard Douglas Powell, M.D.
F. Howard Marsh.
Henry Power.
Sir William MacCormac.
Thomas Pickering Pick.
Charles Arthur Aikin.
1868 H. Charlton Bastian, M.D., F.R.S.
Sir W. H. Broadbent, Bart., M.D.
Thomas Buzzard, M.D.
John Cavafy, M.D.
Walter Butler Cheadle, M.D.
Sir Thos. Crawford, K.C.B., M.D.
T. Henry Green, M.D.
William Chapman Grigg, M.D.
John Croft.
George Eastes.
1869 Joseph Frank Payne, M.D.
Arthur E. Sansom, M.D.
Thomas Laurence Read.
1870 J. Warrington Haward.
Edgcombe Venning.
Clement Godson, M.D.
Reginald Harrison.
Frederick B. Nunneley, M.D.
Robert Leamon Bowles, M.D.
1871 William Cayley, M.D.
Charles Henry Ralfe, M.D.
Thomas L. Brunton, M.D., F.R.S.
J. Hughlings Jackson, M.D., F.R.S.
Henry Sutherland, M.D.
George Vivian Poore, M.D.
Walter Rivington, M.S.
Benjamin Duke.
1872 T. Gilbart Smith, M.D.
George B. Brodie, M.D.
Sir John Williams, Bart., M.D.
Sir J. Fayrer, M.D., F.R.S.
1872 Charles S. Tomes, M.A., F.R.S.
Sir William Bartlett Dalby.
1873 William Miller Ord, M.D.
Frederick Taylor, M.D.
Norman Moore, M.D.
John Curnow, M.D.
William R. Gowers, M.D., F.R.S.
Sir Wm. Guyer Hunter, M.D.
Jeremiah McCarthy.
Wm. Johnson Smith.
Robert William Parker.
Alex. O. MacKellar.
Henry T. Butlin.
Charles Higgens.
William J. Walsham.
1874 Alfred Lewis Galabin, M.D.
George Thin, M.D.
Alfred B. Duffin, M.D.
John Mitchell Bruce, M.D.
Henry Morris.
William Laidlaw Purves.
William Harrison Cripps.
Henry G. Howse, M.S.
Herbert William Page.
Frederic Durham.
William Robert Smith, M.D.
1875 Thomas T. Whipham, M.B.
Francis Charlewood Turner, M.D.
Thomas Crawford Hayes, M.D.
Charles Henry Carter, M.D.
Waren Tay.
Edmund J. Spitta.
Samuel C. Osborn.
Fletcher Beach, M.B.
1876 Thomas Barlow, M.D.
Wm. Lewis Dudley, M.D.
Albert J. Venn, M.D.
John Knowsley Thornton, M.B.
N. Charles Macnamara.
John N. C. Davies-Colley, M.C.
1877 Felix Semon, M.D.
Sidney Coupland, M.D.
Francis Warner, M.D.
William Ewart, M.D.
Alfred Pearce Gould, M.S.
Rickman J. Godlee, M.S.
Alban H. G. Doran.
George Ernest Herman, M.B.
Samuel West, M.D.
John Abercrombie, M.D.
George Allan Heron, M.D.
Joseph A. Ormerod, M.D.
P. Henry Pye-Smith, M.D., F.R.S.
Edward Nettleship.

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1877 William Henry Bennett.
William T. Whitmore.
1878 Sir Jas. Crichton-Browne, M.D.
Fred. T. Roberts, M.D.
Sir Joseph Lister, Bart., F.R.S.
Clinton T. Dent.
John H. Morgan.
Donald W. Charles Hood, M.D.
Henry Gervis, M.D.
Henry Thornton Wharton.
1879 Edward Woakes, M.D.
Armand de Watteville, M.D.
Malcolm A. Morris.
A. E. Cumberbatch.
Edmund Owen.
Arthur E. J. Barker.
Frederick Treves.
Horatio Donkin, M.D.
Thomas John Maclagan, M.D.
Andrew Clark.
Francis Henry Champneys, M.D.
William Watson Cheyne, F.R.S.
George Henry Savage, M.D.
H. H. Clutton, M.A.
Frederic S. Eve.
E. Noble Smith.
William Henry Allchin, M.D.
F. G. Dawtrey Drewitt, M.D.
1880 Robert Alex. Gibbons, M.D.
David Ferrier, M.D., F.R.S.
Vincent Dormer Harris, M.D.
Edmund Distin Maddick.
Jas. John MacWhirter Dunbar, M.D.
James William Browne, M.B.
William Appleton Meredith, M.B.
Alexander Hughes Bennett, M.D.
Malcolm Macdonald McHardy.
A. Boyce Barrow.
William Murrell, M.D.
Leslie Ogilvie, M.B.
George Ogilvie, M.B.
Charles Edward Beevor, M.D.
Thomas Colcott Fox, M.B.
George Henry Makins.
1881 Francis de Havilland Hall, M.D.
Robert Wharry, M.D.
Cecil Yates Biss, M.D.
Richard Clement Lucas, B.S.
Stephen Mackenzie, M.D.
William Hale White, M.D.
Eustace Smith, M.D.
William Sinclair Thomson, M.D.
Percy Kidd, M.D.
Oswald A. Browne.
1881 W. Bruce Clarke, M.B.
Dawson Williams, M.D.
George Lindsay Johnson, M.D.
Henry Edward Juler.
Jonathan F. C. H. Macready.
C. B. Lockwood.
1882 Philip J. Hensley, M.D.
Ernest Clarke, M.D.
John Barclay Scriven.
George Robertson Turner.
Howard Henry Tooth, M.D.
Herbert Isambard Owen, M.D.
Charles R. B. Keetley.
Anthony A. Bowlby.
Amand J. McC. Routh, M.D.
Seymour J. Sharkey, M.D.
William Lang.
Henry Radcliffe Crocker, M.D.
1883 Edwin Clifford Beale, M.A., M.B.
James Kingston Fowler, M.D.
James Frederic Goodhart, M.D.
John Charles Galton, M.A.
W. Hamilton A. Jacobson, M.Ch.
Walter H. Jessop, M.B.
Walter Edmunds, M.C.
Victor A. Horsley, F.R.S.
Dudley Wilmot Buxton, M.D.
Charles Douglas F. Phillips, M.D.
John James Pringle, M.B.
Henry Roxburgh Fuller, M.D.
Wilmot Parker Herringham, M.D.
Augustus Waller, M.D.
William Pasteur, M.D.
Edward Albert Schäfer, F.R.S.
John Bland Sutton.
William Rose, M.B.
Storer Bennett.
Robert Marcus Gunn, M.B.
James Dixon Bradshaw, M.B.
1884 George Newton Pitt, M.D.
Charles Stonham.
Stanley Boyd, M.B.
William Arbuthnot Lane, M.S.
Arthur Marmaduke Sheild, M.B.
Frederic Bowreman Jessett.
Sidney Harris Cox Martin, M.D.
George Lawson.
Thomas Wakley, jun.
F. Swinford Edwards.
James Johnston, M.D.
William Duncan, M.D.
Charles Chinner Fuller.
Jean Samuel Keser, M.D.
George Richard Turner Phillips.

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1884 Bilton Pollard.
Charles E. Jennings, M.D.
1885 Alexander Haig, M.D.
Theodore Dyke Acland, M.D.
Frederick Walker Mott, M.D.
James Berry.
John Cahill.
John Poland.
Heinrich Port, M.D.
R. Norris Wolfenden, M.D.
A. C. Butler-Smythe.
Charles Alfred Ballance, M.S.
Walter S. A. Griffith, M.D.
John Edward Squire, M.D.
John D. Malcolm, M.B., C.M.
Phineas S. Abraham, M.D.
Henry Willingham Gell, M.B.
1886 Robert Maguire, M.D.
Harrington Sainsbury, M.D.
Cuthbert Hilton Golding-Bird, M.B.
Benjamin Wainewright, M.B., C.M.
Charles E. Leopold B. Hudson.
Lauriston Elgie Shaw, M.D.
Charters James Symonds, M.S.
Robert Boxall, M.D.
Allan Ogier Ward, M.D.
Archibald Edward Garrod, M.D.
Stephen Paget.
William Radford Dakin, M.D.
Samuel Herbert Habershon, M.D.
Arthur Quarry Silcock.
Arthur H. N. Lewers, M.D.
1887 Walter George Spencer.
Thomas Outterson Wood, M.D.
Edgar William Willett, M.B.
Henry Lewis Jones, M.D.
Francis George Penrose, M.D.
Hugh Percy Dunn.
Frederic William Hewitt, M.D.
Harry Scott, M.D.
James Barry Ball, M.D.
Gilbert Richardson, M.D.
D'Arcy Power, M.B.
John Gay.
James Calvert, M.D.
Percy J. F. Lush, M.B.
1888 Robert Henry Scanes Spicer, M.D.
Jonathan Hutchinson, jun.
Campbell Williams.
James Donelan, M.B., C.M.
John Anderson, M.D., C.I.E.
Laurie Asher Lawrence.
Charles Arkle, M.D.
Arthur Pearson Luff, M.D., B.Sc.
1888 Albert Carless, M.B., B.S.
Frederick C. Wallis, M.B., B.C.
Charles James Cullingworth, M.D.
Edmund Cautley, M.D., B.C.
H. Montague Murray, M.D.
Arthur Symons Eccles, M.B.
Frank Joseph Wethered, M.D.
Edmund Wilkinson Roughton, M.D.
Frederick William Cock, M.D.
John Phillips, M.B.
1889 Montagu Handfield-Jones, M.D.
Norman M. MacLehose, M.B.
David Henry Goodsall.
Raymond Johnson, M.B.
John Fletcher Little, M.B.
Henry Work Dodd.
George Lindsay Turnbull, M.B.
Sir William Roberts, M.D., F.R.S.
Sidney Phillips, M.D.
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George P. Field.
John Wychenford Washbourn, M.D.
Charles Henry Cosens.
Henry Percy Dean, M.B., M.S.
Alfred Samuel Gubb.
William Hunter, M.D.
J. Inglis Parsons, M.D.
Bernard Pitts, M.B., M.C.
Daniel McClure Ross.
Robert Percy Smith, M.D., B.S.
Herbert R. Spencer, M.D., B.S.
Nestor Isidore Chas. Tirard, M.D.
F. W. Humphery, M.D.
1890 John Rose Bradford, M.D., F.R.S.
Roland Danvers Brinton, M.D.
James Cagney, M.D.
Charles D. B. Hale, M.D.
Edwin Cooper Perry, M.D.
Morton Smale.
Frederick Willcocks, M.D.
R. Ashton Bostock.
William T. Holmes Spicer, M.B.
Thomas Henry Crowle.
Henry Walter Syers, M.D.
Seymour Taylor, M.D.
William Alfred Wills, M.D.
G. O. White-Cooper, M.B.
Herbert William Allingham.
William Anderson.
William A. F. Bateman.
James Jackson Clarke, M.B.
Leonard G. Guthrie, M.B., B.S.
G. William Hill, M.D., B.Sc.
Edward Law, M.D., C.M.

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1891 William Lee Dickinson, M.D.
Herbert P. Hawkins, M.D., B.C.
Cyril Ogle, M.A., M.B.
Leonard Remfry, M.D.
Arthur F. Voelcker, M.D., B.S.
Alfred Pownall Woodforde.
Charles Gordon Brodie.
George Elam.
Herbert T. Herring, M.B., B.S.
Ernest Muirhead Little.
Henry Charrington Martin, M.D.
Launcelot Andrews, M.B.
Frederick William Andrewes, M.B.
Alfred Eddowes, M.D.
Herbert Morley Fletcher, M.D.
William Heaton Hamer, M.D.
William Bromfield Paterson.
Reginald Tweed, M.D.
Holburt Jacob Waring.
Frederic Parkes Weber, M.D.
F. E. Batten, M.B.
Thomas Jessopp Bokenham.
Henry Johnstone Campbell, M.D.
Norman Dalton, M.D.
P. R. W. De Santi.
P. W. Dove.
William J. Gow, M.D.
Charles Arthur Mercier, M.B.
Paul Frank Moline, M.B.
Edward Percy Paton, M.B.
Henry George Read.
Arthur Bowen Rendel, M.B., B.C.
M. Armand Ruffer, M.D.
James Samuel Risien Russell, M.B.
George Cockburn Smith.
Charles Percival White, M.B., B.C.
W. Page May, M.D.
Richard J. Reece.
1892 Edward Cotterell.
J. Dundas Grant, M.D.
R. J. Bliss Howard, M.D.
Thomas Horrocks Openshaw, M.B.
1892 Henry Widenham Maunsell, M.D.
Henry Marmaduke Page.
William Bezly Thorne, M.D.
German Sims Woodhead, M.D.
W .H. Russell Forsbrook, M.D.
John Harold.
William Ward Leadam, M.D.
John Alfred Masters, M.D.
Gustave Schorstein, M.B.
Charles Sempill de Segundo.
John Tweedy.
E. H. Myddelton-Gavey.
E. Matthews James.
J. S. Selwyn-Harvey, M.D.
St. Clair Thomson, M.D.
F. Manley B. Sims.
Solomon Charles Smith, M.D.
F. Poynton Weaver, M.D.
Henry Rayner, M.D.
1893 William Gordon, M.B.
James Taylor, M.D.
Howard Barrett.
Robert Cozens Bailey, M.B.
Henry Albert Caley, M.D.
Arthur Edward Giles, M.D.
Miles Miley, M.B.
George William Davis, M.D.
Alfred A. Kanthack, M.D.
Kenneth McLeod, M.D.
D. Watkin Roberts, M.D.
Leonard A. Bidwell.
Frédéric F. Burghard, M.D, M.S.
William McAdam Eccles, M.B.
Vaughan Harley, M.D.
George Herschell, M.D.
Arnold Lawson.
Walter Knowsley Sibley, M.D.
Richard Sisley, M.D.
Henry Moore Bowman, M.D.
Richard Gill.
Joseph Sefton Sewill.
Thomas Vincent Dickinson, M.D.
Herbert Edward Durham, M.B.
Alexander Morison, M.D.
L. Hemington Pegler, M.D.
Herbt. Furnivall Waterhouse, M.D.
Philip D. Turner, M.D.

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NON-RESIDENT FELLOWS.

Electea

*1851 Acland, Sir Henry W., Bart., K.C.B., M.D., LL.D., F.R.S., Honorary Physician to H.R.H. the Prince of Wales; late Regius Professor of Medicine in the University of Oxford; Oxford.

1839 Alcock, Sir Rutherford, K.C.B., K.C.T., K.T.S., D.C.L., late H.M.'s Envoy Extraordinary at the Court of Pekin. *Trans.* 1.

1866 Allbutt, Thomas Clifford, A.M., M.D., LL.D.Glasgow, F.R.S., Regius Professor of Physic, Univ. Camb.; Consulting Physician to the Leeds General Infirmary; St. Rhadegund's, Cambridge. *Trans.* 3.

1884 Anderson, Alexander Richard, Surgeon to the General Hospital, 5, East Circus Street, Nottingham. *Trans.* 1.

1862 Andrew, James, M.D., Sunnycote, Emery Down, Lyndhurst, Hants. S. 1878-9. C. 1881-2. V.P. 1888. *Trans.* 1.

*1880 Appleton, Henry, M.D., 2, Swinburn villas, Romford, Essex.

1891 Baker, Charles Ernest, M.B., Marlborough road, St. Albans.

*1873 Baker, J. Wright, Consulting Surgeon to the Derbyshire General Infirmary [care of Dr. Benthall, 101, Friar gate, Derby].

1891 Balgarnie, Wilfred, M.B., The Dutch House, Hartley Wintney, Winchfield.

†1848 Ballard, Edward, M.D., F.R.S., late Inspector, Medical Department, Local Government Board; 6, Ravenscroft Park, High Barnet, Herts. C. 1872. V.P. 1875-6. *Sci. Com.* 1889—. *Referee*, 1853-71. *Lib. Com.* 1855. *Trans.* 5.

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*1866 BANKS, SIR JOHN, M.D., LL.D., D.Sc., K.C.B., Physician in Ordinary to the Queen in Ireland; Physician to Richmond, Whitworth, and Hardwicke Hospitals; Consulting Physician to Sir Patrick Dun's and City of Dublin Hospitals; Regius Professor of Physic in the University of Dublin; Member of the Senate of the Royal University in Ireland; 45, Merrion square, Dublin.

1886 BANKS, WILLIAM MITCHELL, M.D., Surgeon to the Liverpool Royal Infirmary; 28, Rodney street, Liverpool.

1882 BARKER, FREDERICK CHARLES, M.D., Surgeon-Major, Bombay Medical Service.

*1881 BARNES, HENRY, M.D., F.R.S. Ed., Physician to the Cumberland Infirmary; 6, Portland square, Carlisle.

†1861 BARNES, ROBERT, M.D., Liss, Hants. C. 1877-8. V.P. 1889–90. *Referee*, 1867-76, 1891—. *Lib. Com.* 1869-73. *Sci. Com.* 1889—. *Trans.* 4.

1840 BARROW, BENJAMIN, Consulting Surgeon to the Royal Isle of Wight Infirmary; Southlands Park road, Ryde.

*1860 BEALEY, ADAM, M.D., M.A., Filsham Lodge, Filsham road, St. Leonard's-on-Sea, Sussex.

1856 BEARDSLEY, AMOS, F.L.S., Bay villa, Grange-over-Sands, Lancashire.

1889 BENTLEY, ARTHUR J. M., M.D., Mena House, Pyramids, Cairo, Egypt.

1872 BEVERLEY, MICHAEL, M.D., Surgeon to the Norfolk and Norwich Hospital; 54, Prince of Wales road, Norwich.

*1865 BICKERSTETH, EDWARD ROBERT, Consulting Surgeon to the Liverpool Royal Infirmary; 2, Rodney street, Liverpool. *Trans.* 1.

1892 BICKERSTETH, ROBERT ALEXANDER, M.A., M.B., Assistant Surgeon to the Liverpool Royal Infirmary; 2, Rodney street, Liverpool.

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1878 BINDON, WILLIAM JOHN VEREKER, M.D., 48, St. Ann's street, Manchester.

†1849 BIRKETT, EDMUND LLOYD, M.D., Consulting Physician to the City of London Hospital for Diseases of the Chest; Westbourne Rectory, Emsworth, Hampshire. C. 1865-6. *Referee*, 1851-9.

1865 BLANCHET, HILARION, Examiner to the College of Physicians and Surgeons, Lower Canada; 35, Conillard street, Quebec, Canada.

1869 BOURNE, WALTER, M.D. (Travelling).

1874 BRADSHAW, A. F., C.B., Surgeon Major-General, Principal Medical Officer, H.M.'s Forces in India; Simla, India. [Agents: Holt & Co., 17, Whitehall place.]

*1867 BRETT, ALFRED T., M.D., Watford House, Watford, Herts.

1876 BRIDGES, ROBERT, M.B., Manor House, Yattendon, Newbury, Berks.

1867 BRIDGEWATER, THOMAS, M.B., LL.D., Harrow-on-the-Hill, Middlesex.

1892 BRONNER, ADOLPH, M.D., 33, Manor row, Bradford.

1893 BROOK, WILLIAM HENRY BREFFIT, 1, James street, Lincoln.

1888 BROWNE, HENRY LANGLEY, Moor House, West Bromwich.

1881 BROWNE, JOHN WALTON, M.D., Surgeon to the Belfast Royal Hospital; Surgeon to the Belfast Ophthalmic Hospital; 10, College square N., Belfast.

1864 BUCKLE, FLEETWOOD, M.D., Merton Lodge, Merton road, Southsea.

1871 BUTT, WILLIAM F.

*1851 CADGE, WILLIAM, Consulting Surgeon to the Norfolk and Norwich Hospital; 49, St. Giles's street, Norwich. *Trans.* 1.

1888 CARTER, WILLIAM JEFFREYS BECHER, Aliwal North, Cape Colony.

1884 CHAFFEY, WAYLAND CHARLES, M.D., Physician to the Royal Alexandra Hospital for Children; 13, Montpellier road, Brighton.

1859 CHANCE, FRANK, M.B., Burleigh House, Sydenham Hill.

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1885 CHAPMAN, PAUL MORGAN, M.D., Physician to the Hereford General Infirmary, 1, St. John street, Hereford. *Trans.* 1.

*1881 CHAVASSE, THOMAS FREDERICK, M.D., C.M., Surgeon to the Birmingham General Hospital; Consulting Surgeon to the Bromsgrove Hospital; 22, Temple row, Birmingham. *Trans.* 3.

1890 CHILDS, CHRISTOPHER, M.D., 2, Royal terrace, Weymouth.

*1873 CHISHOLM, EDWIN, M.D., Abergeldie, Ashfield, near Sydney, New South Wales.

1892 CLARK, JAMES CHARLES, Croft House, Margate road, Southsea.

•1888 CLARKE, ROBERT HENRY, M.B., Westwood, Isle of Thanet, Kent.

1857 COATES, CHARLES, M.D., Consulting Physician to the Bath Royal United Hospital; 10, Circus, Bath.

1868 COCKLE, JOHN, A.M., M.D., F.L.S., Consulting Physician to the Royal Free Hospital; The Lodge, West Molesey. *Trans.* 2.

1893 COLE, ROBERT HENRY, M.B., Moorcroft, Hillingdon, Uxbridge.

1891 COOK, HERBERT GEORGE, M.B., B.S., 22, Newport road, Cardiff.

*1860 CORRY, THOMAS CHARLES STEUART, M.D., Ormeau terrace, and 1, Glenfield place, Belfast.

1891 COUMBE, JOHN BATTEN, M.D., Wargrave, Henley-on-Thames.

*1869 CRESSWELL, PEARSON R., Surgeon to the Merthyr General Hospital; Dowlais, Merthyr Tydvil.

1892 CROSS, FRANCIS RICHARDSON, M.B., Ophthalmic Surgeon to the Bristol Royal Infirmary, and Surgeon to the Bristol Eye Hospital; Worcester House, Clifton, Bristol.

1879 DARWIN, FRANCIS, M.B., F.R.S., Wychfield, Huntingdon road, Cambridge.

1874 DAVIDSON, ALEXANDER, M.D., Physician to the Liverpool Royal Infirmary; 2, Gambier terrace, Liverpool.

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1878 DAVY, RICHARD, Consulting Surgeon to the Westminster Hospital; Burstone House, Bow, North Devon. *Trans.* 1.

*1882 DAWSON, YELVERTON, M.D., Heathlands, Southbourne-on-Sea, Hants.

1889 DELÉPINE, SHERIDAN, B.S., M.B., Professor of Pathology, Owens College, Manchester. *Trans.* 1.

†1839 DIXON, JAMES, Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Harrow Lands, Dorking. L. 1849-55. V.P. 1857-8. T. 1863-4. C. 1866-7. *Referee*, 1865. *Lib. Com.* 1845-8. *Trans.* 4.

1867 DRAGE, CHARLES, M.D., Hatfield, Herts.

1884 DRAGE, LOVELL, M.A., M.B., B.S.Oxon., Burleigh Mead, Hatfield, Herts.

1885 DRUMMOND, DAVID, M.D., 7, Saville place, Newcastle-on-Tyne.

1880 DRURY, CHARLES DENNIS HILL, M.D., Bondgate, Darlington.

1893 DRYSDALE, J. H., 17, Rodney street, Liverpool.

*1871 DUKES, CLEMENT, M.D., B.S., Physician to Rugby School, and Senior Physician to the Hospital of St. Cross, Rugby; Sunnyside, Rugby, Warwickshire.

1867 DUKES, MAJOR CHARLES, M.D., Clarence Villa, Torrs park, Ilfracombe.

*1889 DUNCAN, JOHN, M.D., St. Petersburg, Russia.

1843 DURRANT, CHRISTOPHER MERCER, M.D., Consulting Physician to the East Suffolk and Ipswich Hospital; Northgate street, Ipswich, Suffolk.

1872 EAGER, REGINALD, M.D., Northwoods, near Bristol.

1887 EASMON, JOHN FARRELL, M.D., Assistant Colonial Surgeon, Gold Coast Colony, and Acting Chief Medical Officer of the Colony; Accra, Gold Coast, West Africa.

1887 ELLIOTT, JOHN, Whitefriars Lodge, Chester.

1848 ELLIS, GEORGE VINER, Minsterworth, Gloucester. C. 1863-4. *Trans.* 2.

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1868 ELLIS, JAMES, M.D., the Sanatorium, Anaheim, Los Angeles County, California.

*1854 ELLISON, JAMES, M.D., Surgeon-in-Ordinary to the Royal Household, Windsor; 14, High street, Windsor.

1889 ELLISTON, WILLIAM ALFRED, M.D., Stoke Hall, Ipswich.

*1875 FAGAN, JOHN, Surgeon to, and Lecturer on Clinical Surgery at, the Belfast Royal Hospital; 19, Great Victoria street, Belfast.

1869 FAIRBANK, FREDERICK ROYSTON, M.D., 59, Warrior square, St. Leonard's-on-Sea.

1887 FEENY, MICHAEL HENRY, Les Avants, Montreux, Switzerland.

*1872 FENWICK, JOHN C. J., M.D., Physician to the Durham County Hospital; 25, North road, Durham.

1879 FINLAY, DAVID WHITE, M.D., Professor of the Practice of Medicine in the University of Aberdeen; Physician to the Aberdeen Royal Infirmary; Consulting Physician to the Royal Hospital for Diseases of the Chest, London; 2, Queen's terrace, Aberdeen. *Referee*, 1891—. *Trans.* 2.

1842 FLETCHER, THOMAS BELL ELCOCK, M.D., Consulting Physician to the Birmingham General Hospital; 8, Clarendon crescent, Leamington. *Trans.* 1.

*1864 FOLKER, WILLIAM HENRY, Consulting and late Hon. Surgeon to the North Staffordshire Infirmary; Bedford House, Hanley, Staffordshire.

*1877 DE FONMARTIN, HENRY, M.D., 26, Newberry terrace, Lower Bullar street, Nichol's Town, Southampton.

1892 FOSTER, MICHAEL GEORGE, M.A., M.B., Great Shelford, Cambridge.

1859 FOX, EDWARD LONG, M.D., Consulting Physician to the Bristol Royal Infirmary; Church House, Clifton, Gloucestershire.

1871 FRANK, PHILIP, M.D., Cannes, France.

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*1884 FRANKS, KENDAL, M.D., Surgeon to the Adelaide Hospital and to the Throat and Ear Hospital, Dublin; Surgeon in Ordinary to the Lord Lieutenant; 6, Fitzwilliam square, Dublin. *Trans.* 2.

*1889 FREEMAN, HENRY WILLIAM, 24, The Circus, Bath.

1876 FURNER, WILLOUGHBY, M.D., Surgeon to the Sussex County Hospital; 13, Brunswick square, Brighton.

*1864 GAIRDNER, WILLIAM TENNANT, M.D., LL.D., F.R.S., Physician in Ordinary to H.M. the Queen in Scotland; Professor of the Practice of Medicine in the University of Glasgow; Physician to the Western Infirmary, Glasgow; 225, St. Vincent street, Glasgow. *Trans.* 1.

1885 GAMGEE, ARTHUR, M.D., F.R.S., Davos, Switzerland.

1867 GARLAND, EDWARD CHARLES, Yeovil, Somerset.

1867 GARLIKE, THOMAS W., Malvern Cottage, Churchfield road, Ealing.

1879 GARSTANG, THOMAS WALTER HARROPP, Headingley House, Knutsford, Cheshire.

*1889 GASKELL, WALTER HOLBROOK, M.D., F.R.S., Lecturer on Physiology, University of Cambridge; Petersfield House, Parkside, Cambridge.

1884 GIBBES, HENEAGE, M.D., Professor of Pathology in the University of Michigan; Ann Arbor, Michigan, U.S.A.

1851 GOODFELLOW, STEPHEN JENNINGS, M.D., Consulting Physician to the Middlesex Hospital; Adelphi road, Paignton, Devon. C. 1864-5. *Referee*, 1860-3. *Lib. Com.* 1863. *Trans.* 2.

*1890 GORDON, WILLIAM, M.B., Barnfield Lodge, Exeter.

1889 GREENE, GEORGE EDWARD JOSEPH, Monte Vista, Ferns, County Wexford.

*1875 GREENFIELD, WILLIAM SMITH, M.D., Professor of General Pathology in the University of Edinburgh; 7, Heriot row, Edinburgh. *Sci. Com.* 1879. *Referee*, 1881.

1882 GRESSWELL, DAN ASTLEY, M.A., M.D., D.P.H., Melbourne, Victoria.

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1889 GRIFFITHS, JOSEPH, M.A., M.D., C.M., Assistant to the Professor of Surgery in the University of Cambridge; 17, Fitzwilliam street, Cambridge.

1852 GROVE, JOHN, Pitt House, 15, Johnstown street, Bath.

1870 HAMILTON, ROBERT, Consulting Surgeon to the Royal Southern Hospital, Liverpool; Magheraybuoy, Portrush, Co. Antrim, Ireland.

1892 HARSANT, WILLIAM HENRY, 16, Pembroke road, Clifton, Bristol.

1890 HAVILAND, FRANK PAPILLON, M.B., B.C., 57, Warrior square, St. Leonard's-on-Sea.

1885 HAWKINS, FRANCIS HENRY, M.B., Physician, Royal Berkshire Hospital; 20, Portland place, Reading.

1891 HAYWARD, JOHN ARTHUR, M.B., Chestnuts, Teddington.

1861 HAYWARD, WILLIAM HENRY, Oxford road, Burnley, Lancashire.

†1843 HOLDEN, LUTHER, Consulting Surgeon to St. Bartholomew's Hospital, to the Metropolitan Dispensary, and to the Foundling Hospital; Pinetoft, Ipswich. C. 1859. L. 1865. V.P. 1874. *Referee*, 1866-7. *Lib. Com.* 1858.

1868 HOLLIS, WILLIAM AINSLIE, M.A., M.D., Assistant Physician to the Sussex County Hospital; 8, Cambridge road, Brighton. *Trans.* 1.

†1846 HOLTHOUSE, CARSTEN, 1, Bath terrace, Richmond. C. 1863. *Referee*, 1870-6. *Lib. Com.* 1859-60.

1865 HOWARD, BENJAMIN, M.D. [New York, U.S.] *Trans.* 1.

1881 HOWARD, HENRY, M.B., Medical Officer of Health, Williamstown, Melbourne, Victoria.

1855 HUMPHRY, SIR GEORGE MURRAY, M.D., D.Sc., LL.D., F.R.S., Surgeon to Addenbrooke's Hospital; Professor of Surgery in the University of Cambridge. *Trans.* 9.

1882 HUMPHRY, LAURENCE, M.D., 3, Trinity street, Cambridge.

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1849 HUSSEY, EDWARD LAW, 24, Winchester road, Oxford. *Trans.* 1.

1847 IMAGE, WILLIAM EDMUND, Herringswell House, Mildenhall, Suffolk. *Trans.* 1.

1863 JACKSON, THOMAS VINCENT, Senior Surgeon to the Wolverhampton and Staffordshire General Hospital; Whetstone House, Waterloo road south, Wolverhampton.

*1883 JENKINS, EDWARD JOHNSTONE, M.D., The Australian Club, Sydney, New South Wales.

†1851 JENNER, SIR WILLIAM, Bart., M.D., G.C.B., D.C.L., LL.D.Cantab., LL.D.Edin., F.R.S., Physician in Ordinary to H.M. the Queen, and to H.R.H. the Prince of Wales; Emeritus Professor of Clinical Medicine in University College, London; and Consulting Physician to University College Hospital; Greenwood, Bishop's Waltham, Hants. C. 1864. V.P. 1875. *Referee*, 1855, 1859-63. *Trans.* 3.

1881 JENNINGS, WILLIAM OSCAR, M.D., 35, Rue Marbœuf, Avenue des Champs-Elysées, Paris.

1851 JOHNSON, EDMUND CHARLES, Corresponding Member of the Medical and Philosophical Society of Florence, and of "l'Institut Génevois."

1889 JOHNSON, HAROLD J., Senior Assistant, Gloucester County Asylum.

1848 JOHNSTONE, ATHOL ARCHIBALD WOOD, Consulting Surgeon to the Royal Alexandra Hospital for Sick Children, St. Moritz House, 61, Dyke road, Brighton. *Lib. Com.* 1860. *Trans.* 1.

1876 JONES, LESLIE HUDSON, M.D., Limefield House, Cheetham hill, Manchester.

*1875 JONES, PHILIP SYDNEY, M.D., Consulting Surgeon to the Sydney Infirmary; Examiner in Medicine, and Fellow of the Senate, Sydney University; 10, College street, Sydney, New South Wales. [Agents: Messrs. D. Jones & Co., Wool Exchange.]

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1865 JORDAN, FURNEAUX, Consulting Surgeon to the Queen's Hospital, Birmingham; Selly Hill, Birmingham.

1872 KELLY, CHARLES, M.D., Professor of Hygiene in King's College, London, and Medical Officer of Health for the West Sussex Combined Sanitary District; Ellesmere, Gratwicke road, Worthing, Sussex.

*1848 KENDELL, DANIEL BURTON, M.B., Thornhill House, Walton, near Wakefield, Yorkshire.

*1890 KERR, J. G. DOUGLAS, M.B., C.M., 6, The Circus, Bath.

*1877 KHORY, RUSTOMJEE NASERWANJEE, M.D., Honorary Obstetric Physician to the Bai Motlibari and Sir Dinsha Petil Hospitals, &c.; Hormazd Villa, Khumballa hill, Bombay.

1883 KNAPTON, GEORGE, Queen's square, Blackpool.

1888 KYNSEY, WILLIAM RAYMOND, C.M.G., Inspector-General of Hospitals, Colombo, Ceylon.

1889 LANCASTER, ERNEST LE CRONIER, M.B., B.Ch., Assistant Physician to the Swansea Hospital; Winchester House, Swansea, S. Wales.

1891 LANE, HUGH, 11, The Circus, Bath.

*1873 LARCHER, O., M.D., Laureate of the Institute of France, of the Medical Faculty, and Academy of Paris, &c.; 97, Rue de Passy, Passy, Paris.

1862 LATHAM, PETER WALLWORK, M.A., M.D., Downing Professor of Medicine, Cambridge University; Senior Physician to Addenbrooke's Hospital, Cambridge; 17, Trumpington street, Cambridge.

*1890 LAWRIE, EDWARD, M.B., Surgeon Lieutenant-Colonel, Indian Medical Department; Residency Surgeon; Hyderabad, Deccan.

1880 LAYCOCK, GEORGE LOCKWOOD, M.B., C.M., Melbourne, Victoria, Australia.

1892 LAZARUS-BARLOW, WALTER SYDNEY, M.B., The Acacias, Chesterton, Cambridge. *Sci. Com.* 1892—.

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*1886 LEDIARD, HENRY AMBROSE, M.D., Surgeon to the Cumberland Infirmary; 35, Lowther street, Carlisle.

1882 LEDWICH, EDWARD L'ESTRANGE, Anatomist to the Royal College of Surgeons, Ireland; 31, Harcourt street, Dublin.

1883 LEESON, JOHN RUDD, M.D., C.M., Clifden House, Twickenham.

1869 LEGG, JOHN WICKHAM, M.D., C. 1886. *Referee*, 1882-5 *Lib. Com.* 1878-85. *Trans.* 2.

1872 LIEBREICH, RICHARD (Consulting Ophthalmic Surgeon to St. Thomas's Hospital, London); Paris.

*1872 LITTLE, DAVID, M.D., Senior Surgeon to the Royal Eye Hospital, Manchester; Ophthalmic Surgeon to the Manchester Royal Infirmary; Lecturer on Ophthalmology at the Victoria University; 21, St. John street, Manchester.

*1889 LITTLE, JAMES, M.D., Physician to the Adelaide Hospital; Consulting Physician to the Rotunda, St. Mark's, Steevens' and the Children's Hospitals; 14, Stephen's Green North, Dublin.

1871 LITTLE, LOUIS STROMEYER, Shanghai, China.

1860 LONGMORE, SIR THOMAS, C.B., Hon. Surgeon to H.M. the Queen; Surgeon-General, Army Medical Staff (Retired); Foreign Corresponding Member, Académie de Médecine; Assoc. Soc. Chir. de Paris; Officer of Legion of Honour; The Paddock, Woolston, Hants. *Trans.* 2.

1883 LUND, EDWARD, Emeritus Professor of Surgery, Victoria University, Manchester; Consulting Surgeon to the Manchester Royal Infirmary; 22, St. John street, Manchester.

1867 MABERLY, GEORGE FREDERICK, Mailai Valley, Nelson, New Zealand.

1889 MACALISTER, DONALD, M.A., B.Sc., M.D., Physician to Addenbrooke's Hospital; Lecturer on Medicine, St. John's College; University Lecturer in Medicine; St. John's College, Cambridge.

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Elected

1887 MACDONALD, GEORGE CHILDS, M.D.

1866 MACGOWAN, ALEXANDER THORBURN, M.D.

*1859 M'INTYRE, JOHN, M.D., LL.D., Odiham, Hants.

1876 MACKEY, EDWARD, M.D., Senior Physician to the Royal Alexandra Hospital for Sick Children; Assistant Physician to the Sussex County Hospital; 3, Portland place, Brighton.

*1854 MACKINDER, DRAPER, M.D., Consulting Surgeon to the Dispensary, The Cedars, Gainsborough, Lincolnshire.

1891 MANBY, ALAN REEVE, M.D., Surgeon Apothecary to their Royal Highnesses the Prince and Princess of Wales at Sandringham; East Rudham, Norfolk.

1892 MARTIN, CHRISTOPHER, M.B., C.M., 35, George road, Edgbaston, Birmingham.

1883 MAUDSLEY, HENRY CARR, M.D., 22, Collins street, Melbourne, Victoria.

1839 MEADE, RICHARD HENRY, Consulting Surgeon to the Bradford Infirmary; Bradford, Yorkshire. *Trans.* 1.

1874 MERRIMAN, JOHN J., 13, The Steyne, Worthing.

1882 MILLS, JOSEPH, Claremont North, Andover, Hants.

1887 MIVART, FREDERICK ST. GEORGE, M.D., Beaumont Lodge, Worple road, Wimbledon.

1883 MONEY, ANGEL, M.D., Sydney, New South Wales.

1893 MOONEY, JOSEPH JOHN, 35, Stretford road, Manchester.

1891 MORRIS, GRAHAM, Wallington, Surrey.

1893 MORSE, THOMAS HERBERT, 10, Upper Surrey street, Norwich. *Trans.* 1.

1873 MURRAY, J. IVOR, M.D., 24, Huntriss row, Scarborough.

1889 NAPIER, FRANCIS HORATIO, M.B., Cape Town.

1881 NALL, SAMUEL, M.B., Dryhurst Lodge, Disley, Stockport.

1870 NEILD, JAMES EDWARD, M.D., Lecturer on Forensic Medicine and Psychological Medicine in the University of Melbourne; 21, Spring street, Melbourne, Victoria.

1868 NICHOLLS, JAMES, M.D., Trenarren, Newquay, Cornwall.

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Elected

1849 NORMAN, HENRY BURFORD, The Manor House, Drayton, Taunton, Somerset. *Lib. Com.* 1857.

*1847 NOURSE, WILLIAM EDWARD CHARLES, Bouverie House, Exeter.

1884 OAKES, ARTHUR, M.D., Warialda, Portarlington Road, Bournemouth.

1880 O'CONNOR, BERNARD, A.B., M.D., Physician to the North London Hospital for Consumption; 25, Hamilton road, Ealing.

1847 O'CONNOR, THOMAS, March, Cambridgeshire.

*1855 OGLE, WILLIAM, M.A., M.D., late Physician to the Derbyshire Infirmary; The Elms, Duffield road, Derby.

1870 OLDHAM, CHARLES FREDERIC, India [Agents: Messrs. Grindlay and Co., 55, Parliament street].

*1883 OLIVER, THOMAS, M.A., M.D., Professor of Physiology, University of Durham; and Physician to the Newcastle-on-Tyne Infirmary; 7, Ellison place, Newcastle-on-Tyne. *Trans.* 1.

*1871 O'NEILL, WILLIAM, M.D., late Physician to the Lincoln Lunatic Hospital, 2, Lindum road, Lincoln.

1885 ORMSBY, L. HEPENSTAL, M.D., Lecturer on Clinical and Operative Surgery and Surgeon to the Meath Hospital and County Dublin Infirmary; Surgeon to the Children's Hospital, Dublin; 92, Merrion square west, Dublin.

1887 PAGET, CHARLES EDWARD, Medical Officer of Health for the County Borough of Salford; North Bentcliffe, Eccles, Lancashire.

*1858 PALEY, WILLIAM, M.D., Physician to the Ripon Dispensary; Yore Bank, Ripon, Yorkshire.

1887 PARDINGTON, GEORGE LUCAS, M.D., 47, Mount Pleasant road, Tunbridge Wells.

1885 PARKER, RUSHTON, M.B., B.S., Professor of Surgery, University College, Liverpool (Victoria University); Surgeon to the Liverpool Royal Infirmary; 59, Rodney street, Liverpool.

1891 PARKIN, ALFRED, M.S., M.D., 5, Albion street, Hull. *Trans.* 1.

1879 PEEL, ROBERT, 120, Collins street east, Melbourne, Victoria.

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1856 PEIRCE, RICHARD KING, Laggan House, Maidenhead.

*1855 PEMBERTON, OLIVER, Consulting Surgeon to the Birmingham General Hospital, and Professor of Surgery at the Queen's College, Birmingham; H.M. Coroner, Birmingham; 65, Temple row, Birmingham. *Trans.* 1.

1874 PENHALL, JOHN THOMAS, The Cedars, Broadwas-on-Teme, Worcester.

*1879 PESIKAKA, HORMASJI DOSABHAI, late Hon. Surgeon to the G. T. Hospital (Bombay); 43, Hornby Road, Bombay.

*1878 PHILIPSON, GEORGE HARE, M.D., M.A., D.C.L., Professor of Medicine at Durham University; Senior Physician to the Newcastle-upon-Tyne Royal Infirmary; 7, Eldon square, Newcastle-upon-Tyne.

1891 PIERCE, BEDFORD, M.D., The Retreat, York.

†1841 PITMAN, SIR HENRY ALFRED, M.D., Consulting Physician to St. George's Hospital; Cranbrook, Bycullah park, Enfield. L. 1851-3. C. 1861-2. T. 1863-8. V.P. 1870-1. *Referee*, 1849-50. *Lib. Com.* 1847.

1892 POWELL, HERBERT ANDREWS, M.A., M.D., M.Ch., 9, St. Thomas's street, Winchester.

1857 VON RANKE, HENRY, M.D., 3, Sophienstrasse, Munich.

1893 RANKIN, GUTHRIE, M.D., 23, Jury street, Warwick.

1890 RANSOM, WILLIAM BRAMWELL, M.D., Physician to the Nottingham General Hospital; The Pavement, Nottingham. *Trans.* 1.

1854 RANSOM, WILLIAM HENRY, M.D., F.R.S., Consulting Physician to the Nottingham General Hospital; The Pavement, Nottingham. *Trans.* 1.

1882 REID, JAMES, M.D., C.B., Resident Physician, and Physician in Ordinary to H.M. the Queen, Windsor Castle.

1884 REID, THOMAS WHITEHEAD, Surgeon to the Kent and Canterbury Hospital; St. George's House, Canterbury, Kent.

1881 RICE, GEORGE, M.B., C.M., Sutton, Surrey.

1889 RIVERS, W. H. RIVERS, M.D., St. John's College, Cambridge.

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*1871 ROBERTS, DAVID LLOYD, M.D., Obstetric Physician to the Manchester Royal Infirmary; Physician to St. Mary's Hospital, and Lecturer on Clinical Obstetrics and Gynæcology at the Owens College, Manchester; 11, St. John street, Manchester.

1889 ROBERTS, H. LESLIE, M.B., C.M., 31, Rodney street, Liverpool.

1873 ROBERTSON, WILLIAM HENRY, M.D., Consulting Physician to the Buxton Bath Charity and Devonshire Hospital Buxton, Derbyshire.

*1888 ROBINSON, FREDERICK WILLIAM, M.D., C.M., Huddersfield.

1889 ROBSON, ARTHUR WILLIAM MAYO, 7, Park square, Leeds. *Trans.* 2.

1885 ROCKWOOD, WILLIAM GABRIEL, M.D., Colombo, Ceylon.

1850 ROPER, GEORGE, M.D., Consulting Physician to the Eastern Division of the Royal Maternity Charity; and to the Royal Infirmary for Children and Women, Waterloo Bridge road; Oulton Lodge, Aylsham, Norfolk. C. 1879-80.

1863 ROWE, THOMAS SMITH, M.D., Consulting Surgeon to the Royal Sea-Bathing Infirmary; Union crescent, Margate, Kent.

1882 ROY, CHARLES SMART, M.D., F.R.S., Professor of Pathology in the University of Cambridge; Trinity College, Cambridge.

1871 RUTHERFORD, WILLIAM, M.D., F.R.S., Professor of the Institutes of Medicine in the University of Edinburgh; 14, Douglas crescent, Edinburgh.

1856 SALTER, S. JAMES A., M.B., F.R.S., F.L.S., Basingfield, near Basingstoke, Hants. C. 1871. *Lib. Com.* 1878. *Trans.* 2.

†1855 SANDERSON, JOHN BURDON, M.D., LL.D., D.C.L. Durham, D.Sc., F.R.S., Waynflete Professor of Physiology in the University of Oxford; 64, Banbury road, Oxford. C. 1869-70. V.P. 1882. *Referee*, 1867-8, 1876-81. *Sci. Com.* 1862, 1870. *Lib. Com.* 1876-81. *Trans.* 2.

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1867 SANDFORD, FOLLIOTT JAMES, M.D., Surgeon-Major, Medical Officer of Health of the Drayton Union Rural Sanitary District; Surgeon to the Market Drayton Dispensary; and Consulting Physician to the Market Drayton Cottage Hospital; Market Drayton, Shropshire.

1886 SAUNDBY, ROBERT, M.D., Physician to the General Hospital, and Consulting Physician to the Hospital for Women, and to the Eye Hospital, Birmingham; Professor of Medicine, Mason College; 83A, Edmund street, Birmingham.

1891 SAUNDERS, FREDERICK WILLIAM, M.B., B.C., Chieveley House, near Newbury, Berks.

*1861 SCOTT, WILLIAM, M.D., Senior Physician to the Huddersfield Infirmary; Waverley House, Huddersfield.

1840 SHARP, WILLIAM, M.D., F.R.S., Horton House, Rugby. *Trans.* 1.

1887 SIDEBOTHAM, EDWARD JOHN, M.B., Erlesdene, Bowdon, Cheshire.

1857 SIORDET, JAMES LEWIS, M.D., Villa Labrolles, Mentone, Alpes Maritimes, France.

1886 SMITH, HOWARD LYON, Buckland House, Buckland Newton, Dorchester.

1885 SMITH, JAMES GREIG, M.B., C.M., Lecturer on Surgery, Bristol Medical School; Surgeon to the Bristol Royal Infirmary; 16, Victoria square, Clifton, Bristol. *Trans.* 1.

1893 SMITH, ROBERT SHINGLETON, M.D., Deepholm, Clifton Park, Clifton, Bristol.

1868 SOLLY, SAMUEL EDWIN, Colorado Springs, Colorado, U.S.

1891 STEVENS, CECIL ROBERT, M.B., B.S., Marwood House, Honiton, Devon.

1854 STEVENS, HENRY, M.D., late Inspector, Medical Department, Local Government Board, Whitehall; Falcon Lodge, Hampton, Middlesex.

1884 STEWART, EDWARD, M.D., Brook House, East Grinstead.

*1879 STIRLING, EDWARD CHARLES, M.A., M.D., Senior Surgeon to the Adelaide Hospital; Lecturer on Physiology in the University of Adelaide, South Australia [care of Messrs. Elder and Co., 7, St. Helen's place].

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1865 STOKES, SIR WILLIAM, M.D., M.C., Surgeon to the Meath Hospital; 5, Merrion square north, Dublin. *Trans.* 1.

1871 STRONG, HENRY JOHN, M.D., Consulting Surgeon to the Croydon General Hospital; Colonnade House, The Steyne, Worthing.

*1890 SYMPSON, E. MANSEL, M.A., M.D., B.C., 3, James street, Lincoln.

1870 TAIT, LAWSON, Surgeon to the Birmingham and Midland Hospital for Women; 7, The Crescent, Birmingham. *Trans.* 6.

1886 TEALE, THOMAS PRIDGIN, M.B., F.R.S., Consulting Surgeon to the Leeds General Infirmary; 38, Cookridge street, Leeds.

1890 THOMAS, WILLIAM ROBERT, M.D., Little Forest, Bath road, Bournemouth.

1891 THOMSON, JOHN ROBERTS, M.D., Monkchester, Bournemouth.

1883 THURSFIELD, THOMAS WILLIAM, M.D., Physician to the Warneford and South Warwickshire General Hospital; Selwood, Beauchamp square, Leamington.

1880 TIVY, WILLIAM JAMES, 8, Lansdowne place, Clifton, Bristol.

*1871 TREND, THEOPHILUS W., M.D., Physician to Royal South Hants Infirmary; 1, Grosvenor square, Southampton.

*1881 TREVES, WILLIAM KNIGHT, Surgeon to the National Hospital for Scrofula; 31, Dalby square, Cliftonville, Margate.

1867 TROTTER, JOHN WILLIAM, formerly Surgeon-Major, Coldstream Guards; 4, St. Peter's terrace, York.

1873 TURNER, GEORGE BROWN, M.D., The Lodge, Hemel Hempsted, Herts.

1881 TYSON, WILLIAM JOSEPH, M.D., Medical Officer of the Folkestone Infirmary; 10, Langhorne Gardens, Folkestone.

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1828 VULPES, BENEDETTO, M.D., Physician to the Hospital of Aversa, and the Hospital of Incurables, Naples.

1854 WADDINGTON, EDWARD, Hamilton, Auckland, New Zealand.

*1868 WALKER, ROBERT, Clovelly, Bideford.

1887 WALLACE, EDWARD JAMES, M.D., Holmbush, Grove road, Southsea.

1867 WALLIS, GEORGE, Consulting Surgeon to Addenbrooke's Hospital; 6, Hills road, Cambridge.

*1883 WALTERS, JAMES HOPKINS, Surgeon to the Royal Berkshire Hospital; 15, Friar street, Reading.

1821 WARD, WILLIAM TILLEARD, Tilleards, Stanhope, Canada.

1893 WARD-HUMPHREYS, GEORGE HERBERT, Oriel Lodge, Cheltenham.

1846 WARE, JAMES THOMAS, Tilford House, near Farnham, Surrey.

1861 WATERS, A. T. HOUGHTON, M.D., Consulting Physician to the Royal Infirmary; 69, Bedford street, Liverpool. *Trans.* 3.

1840 WEBB, WILLIAM WOODHAM, M.D., Neuilly-sur-Seine, France.

1874 WELLS, HARRY, M.D., San Ysidro, Buenos Ayres, S. America.

1882 WHARRY, CHARLES JOHN, M.D., 14, Ewell road, Surbiton, Surrey.

*1881 WHITEHEAD, WALTER, F.R.S. Ed., Senior Surgeon to the Manchester Royal Infirmary, Manchester and Salford Lock Hospital, and Manchester and Salford Skin Hospital; 499, Oxford road, Manchester. *Trans.* 1.

1885 WHITLA, WILLIAM, M.D., Professor of Materia Medica and Therapeutics, Queen's College, Belfast; Physician to, and Lecturer in Medicine at, the Belfast Royal Hospital; Consulting Physician to the Ulster Hospital for Women and Children; 8, College square north, Belfast.

1852 WIBLIN, JOHN, The Hermitage, Clewer, Windsor. *Trans.* 1.

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*1870 WILKIN, JOHN F., M.D., M.C., The Warren, Beckenham, Kent.

*1883 WILKINSON, THOMAS MARSHALL, late Surgeon to the Lincoln County Hospital and to the Lincoln General Dispensary; 33, Avenue road, Grantham.

1837 WILKS, GEORGE AUGUSTUS FREDERICK, M.D., Stanbury, Torquay.

*1883 WILLANS, WILLIAM BLUNDELL, Much Hadham, Herts.

*1859 WILLIAMS, CHARLES, Senior Surgeon to the Norfolk and Norwich Hospital; 48, Prince of Wales road, Norwich.

1887 WILSON, ARTHUR HERVEY, M.D., 504, Broadway, Boston, U.S.A.

1863 WILSON, ROBERT JAMES, 7, Warrior square, St. Leonard's-on-Sea, Sussex.

1889 WISE, A. TUCKER, M.D., Davos Platz, Switzerland.

*1850 WISE, ROBERT STANTON, M.D., Consulting Physician to the Southam Eye and Ear Infirmary; Beech Lawn, Banbury.

1883 WOOD, WILLIAM EDWARD RAMSDEN, M.A., M.D., The Priory, Roehampton.

1879 WOODWARD, G. P. M., M.D., Deputy Surgeon-General; 157, Liverpool street, Hyde park, Sydney, New South Wales.

1892 WRIGHT, ALMROTH EDWARD, M.D., Ch.B., Oakhurst, Netley, Hants.

[It is particularly requested that any change of Title, Appointment, or Residence, may be communicated to the Hon. Secretaries before the 1st of September in each year, in order that the List may be made as correct as possible.]

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ANNUAL GENERAL MEETING.

Thursday, March 1st, 1894.

Dr. WILLIAM SELBY CHURCH, M.D., Senior Vice-President, in the Chair.

J. MITCHELL BRUCE, M.D., RICKMAN J. GODLEE, M.S., } Hon. Secs.

The Minutes of the last Annual General Meeting were read and confirmed.

The Chairman nominated as Scrutineers Dr. H. A. Caley and Dr. George Knapton, and requested them to superintend the Ballot.

The Chairman then called upon the Senior Hon. Secretary, Mr. Rickman J. Godlee, to read the

REPORT OF THE COUNCIL.

In presenting its Report the Council has, with profound regret, to record the loss which it has sustained by the death of Sir Andrew Clark. The accurate knowledge that he possessed of all the details of the Society's work was such that in each department of it his loss has

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been severely felt. This is the first time in the history of the Society that a President has died during his term of office, and the Council was, therefore, without a precedent to guide it in deciding upon the best course to pursue under the circumstances. It was determined to recommend that the Senior Medical Vice-President, Dr. Church, should discharge the duties of President until the next Annual General Meeting, and the recommendation was accepted by the Society.

The House Committee, which grew out of the Building Committee appointed to supervise the alterations of the Society's house, has been enlarged and brought into more immediate relation with the Council. It now consists of the President, the Senior Treasurer, and the Secretaries, *ex officio*, and of five other Members, one of whom only is a Member of Council. Upon the House Committee devolves a large part of the business transactions of the Society, which had increased so much that they seriously hampered the work of the Council. All matters connected with tenancies, letting, repairs, and service, besides other questions specially referred to it by the Council, are now determined by this body. This arrangement has greatly lightened the work of the Council.

The financial position of the Society has been under the very serious consideration of the Council during the last Session. A committee was appointed to investigate the subject, and many meetings were held. Various suggestions were made by which, if it had been thought advisable, the income of the Society might have been increased. None of these plans were adopted, but certain measures of economy were carried out which at the same time were obviously conducive to the advantage of the Society. The most important of these were (1) an alteration in the method of issuing the 'Proceedings;' and (2) the abandonment of the plan of producing the electric current on the premises, and the letting on lease of the cellar formerly occupied by the electric plant. The conclusion having been reached that the financial

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position is sound, further changes were considered to be uncalled for.

On the death of Sir Andrew Clark, a vacancy occurred in the office of Trustee. Dr. Frederick Taylor was selected by the Council for this appointment.

The interest in the Society's meetings has not diminished, but, on the contrary, seems to have increased, as evidenced by the number and value of the papers presented, by the large attendance at the meetings, and by the high standard of the discussions.

One of the last acts done by the late President for the benefit of the Society was the confirmation of an advantageous arrangement that had been entered into with Messrs. Macmillan, by which they undertook, at their own expense, to publish the voluminous report of the Committee upon the Medical Climatology and Balneology of the United Kingdom. In reference to the work of this Committee the following statement has been received from its Honorary Secretary:

> "The Committee on the Climatology and Balneology of the British Isles has presented a portion of their report to the Council. Arrangements have been made with Messrs. Macmillan for the publication of the report.
>
> "The part dealing with the Climatology of the South Coast is being pressed on for publication, and is now practically complete, and the portion dealing with Balneology will also be completed shortly."

The following is the Report of the Committee on Suspended Animation:

> "The Committee appointed to investigate the subject of Suspended Animation in the Drowned has held several meetings, and has performed experiments on the dead body. They are not in a

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position yet to make any report to the Society. Their investigations have been delayed by the difficulties they have experienced in obtaining suitable subjects for experiment."

The following is the Report of the Honorary Librarians:

"We record with sincere regret the loss which the Society has sustained by the death of an old and esteemed servant, viz. Mr. Richard Coldrey, who entered the service of the Society as a boy forty-five years ago, and by his faithfulness and devotion to the interests of the Society earned for himself the regard and esteem, not only of the officers of the Society, but of all Fellows who came to know him.

"We feel sure that it will be very gratifying to the body of the Fellows that the subscription which was started by the late President is likely to result in a substantial sum being placed at the service of Mr. Coldrey's family.

"Mr. Coldrey's death made a desirable rearrangement of the work of the Society possible. A not inconsiderable amount of the Resident Librarian's working time and energy is engrossed by business not directly connected with the Library and with the Society's publications, a circumstance incidental to the imposition upon him of new and multifarious duties arising out of the occupancy of the Society's new house. This, together with recurrent interruptions caused by the receipt of subscriptions to this and also to other medical societies using these premises, having been recognised as prejudicial to thorough efficiency in relation to library work, it was decided to divide the duties, and Mr. Archibald Clarke, formerly Librarian at the Middlesex Hospital, was

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appointed Sub-librarian in the place of Mr. Coldrey, and his duties were limited to work in the library. At the same time Mr. H. H. Swinny was appointed accountant, his duties being solely confined to financial matters. We hope that this arrangement will largely conduce to the convenience of the Fellows using the Library, and to relieve from non-scientific work the Resident Librarian, whose thorough devotion to the interests of the Society is beyond doubt.

"There are no additions of any exceptional value to report, but the supply of books has been steadily kept up. An increased subscription has been arranged for with Lewis's Library, which secures for the use of the Fellows thirty new volumes at a time, and enables the Librarian by rapid exchanges to obtain any work which may be required at a few days' notice.

"The sum of £200 has been expended upon additions to the Library, and £30 on binding; and many valuable gifts have been received."

SAMUEL GEE, J. W. HULKE, } *Hon. Librarians.*

The following is the Treasurers' Report:

"The total receipts of the Society for the year 1893 from its normal sources of income were £4052 14*s.* 1*d.*, and its payments on current account were £3883 9*s.* 3*d.* This latter sum included some disbursements of an unusual kind, such as that for the extra help required in consequence of the long illness of the Sub-Librarian, Mr. Coldrey, and more than the average expenditure under the head of lighting, heating, &c. Still there would, notwithstand-

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ing these unusual payments, have remained an excess of receipts over payments of £169 4*s*. 10*d*. had it not been that we have during the past year paid off several rather considerable items connected with the structural alterations of the premises, the cost of which correctly belonged to other years, and, had the Debenture Capital been large enough, would, of course, have been paid out of it. The capital amount thus paid in 1893 was £289 16*s*. 9*d*., but this was partially met by the sale of our electric plant (no longer needed, as we are now getting the electric current more economically from one of the Supply Companies), which produced £180 11*s*. 6*d*. The net result is, therefore, that our total receipts for 1893 have exceeded our payments by £59 19*s*. 7*d*.

"The annual subscriptions in 1892 reached £1272 12*s*., a larger amount than they had ever before attained; but during the past year they amounted to £1367 2*s*., a still further increase in this most important branch of our income of over £94. Again, the rental of our premises in 1893 was more than £90 in excess of that of the preceding year, and amounted exactly to £2461 16*s*. 6*d*."

The Council cannot close its Report without acknowledging its indebtedness to Dr. Hare, the Senior Treasurer, now retiring from office. During the most anxious financial period in the history of the Society Dr. Hare devoted himself to the duties of his office with a zeal and assiduity which it is impossible to exaggerate.

The roll of the Society now contains 500 Resident, 269 Non-resident, and 19 Honorary Fellows, or a total of 788.

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The Chairman then called upon Dr. Hare, the Senior Treasurer, to read the Annual Statement of Accounts (see p. xcii).

In introducing the accounts to the notice of the Fellows Dr. Hare explained the financial position of the Society, and observed that although it was only fair to the officials of the Society that two or three days' notice should be given of important questions, he was nevertheless quite willing as far as possible to reply to any questions that Fellows might put then and there. The Report on the whole was of a highly satisfactory character. During the past year no composition fees had been paid, a circumstance upon which, speaking as a Treasurer, he thought the Society was to be congratulated. Under the heading of firing he called attention to the fact that, thanks to the foresight of the Resident Librarian, the Society had not suffered in the least from the coal famine which had marked the past winter, a sufficient supply having been laid in during last summer to provide for their needs. A very large sum comparatively had been spent on the hire of microscopes during the year 1892, but it had been found possible to reduce this sum very materially during the last financial year. Referring to the sums spent on the room vacated by the Society for the Relief of Widows and Orphans of Medical Men, he pointed out that arrangements had been made by which that room would in future bring in a rent of £120 per annum in lieu of £50 as heretofore. He concluded by moving the reception and adoption of the Report and Statement of Accounts.

Dr. Charles West observed that after the highly satisfactory account given of the present position of the Society by the Council, and the encouraging and lucid explanations given by Dr. Hare, there only remained to him to second the adoption of the report, &c. He could not do so, however, without congratulating them on their progress in every way,—not merely their financial progress, though this was indisputably of great importance, but also on

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their unremitting labours for the advancement of medical science. The progress of the Society had been excellent all along the line; they had been gathering strength as they went along, and he trusted it might continue to be the same through all time,—in fact, that their success might be as perpetual as anything in this changing world could well be.

Dr. THEODORE WILLIAMS asked whether they had begun to pay off the debenture-holders.

Dr. HARE, in reply, said that under the deed the Society could not do so until after January 1st next. They would do so then unless the trustees thought proper to relieve them from so doing.

The CHAIRMAN then moved, and Dr. CHARLES WEST seconded—"That the Report of the Council, together with the Treasurers' audited Statement of Accounts, be adopted, and printed in the next volume of 'Transactions.'"

This was carried unanimously.

The CHAIRMAN then proceeded to deliver the

ANNUAL ADDRESS (p. 1).

Sir EDWARD SIEVEKING moved—"That the best thanks of the Society be given to the Senior Vice-President for his valuable and important services and for the Address just delivered, and that he be requested to allow the Address to be printed in the next volume of the 'Transactions.'" In doing so he alluded to the very interesting and successful career of their late President, Sir Andrew Clark, observing that one could hardly wish for a more desirable ending to a prosperous career, quitting life, as their President had done, in the height of his fame. He thanked Dr. Church for having given them so many new and interesting details about their late President's career. He expressed great satisfaction at the announcement that the Balneological Committee was now about to make a first report. He had been looking forward anxiously for this report, and he would welcome it. The idea in the minds of those who first started this committee on its

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way was that this step might pave the way to and facilitate the formation of a "Royal Academy of Medicine." He hoped that the scheme of standing committees would be adopted, particularly in relation to balneology, because that was a subject which practically could never come to an end. He hoped that not only this but other committees would be appointed, to which medical men could refer. He hoped, too, that the time would come when the Society would occupy and utilise its premises instead of being merely the lessor of the greater part. He apologised for introducing these topics, but said that they were matters on which he had felt very strongly for years.

Dr. KENNETH MACLEOD said that having been present at most of the meetings since Dr. Church had occupied the presidential chair, he was personally in a position to testify to his grace and urbanity in the discharge of his functions. He concluded by seconding the motion.

This was carried by acclamation.

The Senior Vice-President, Dr. CHURCH, thanked the Fellows for the kind way in which they had recognised whatever services he had been enabled to render, but he pointed out that he himself lacked the experience of the post, and the credit, whatever it might be, was properly that of the officers of the Society who had so cordially assisted him with their advice and experience.

Mr. A. WILLETT then moved—"That the best thanks of the Society be given to the retiring Vice-Presidents, Dr. Church and Mr. Power, for their services to the Society during their term of office."

Dr. SANSOM, in seconding the motion, said it was somewhat painful at such meetings of the Society that they should have to listen to the *morituri te salutant* of departing officers, but that was, he supposed, inevitable. He regretted that Sir William Priestley, who had intended to support this resolution, had been compelled to leave without doing so, and humorously remarked on the association of "Church and Power" which

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narrowly escaped being brought about by "Priestley" influence.

Dr. CHURCH briefly acknowledged the thanks on behalf of himself and of Mr. Power.

Dr. G. VIVIAN POORE moved—"That the most cordial thanks of the Society be given to the retiring Senior Treasurer, Dr. Hare, for his most important and valuable services to the Society during a most critical period in the history of the Society." He said during the Presidency of Mr. Holmes the Society had passed, so to speak, through a crisis, and he himself knew something of the enormous amount of labour that devolved upon the Treasurer at that time. The explanation which Dr. Hare had just given them showed that he was no nominal treasurer, but had himself audited his accounts and had taken the greatest interest in them. He said he would introduce one amendment into the motion as it stood, viz. that Dr. Hare's servces, instead of being described as "valuable," should be described as "invaluable."

This was seconded by Mr. F. DURHAM, and carried by acclamation.

Dr. HARE said he had felt it a great honour to have been appointed one of the Treasurers of the Society, though he had little idea of the amount of work that this post would involve. Still that very work had associated him still more closely with the Society, and had made him feel a deeper interest in its affairs. He rejoiced also at the fact that it had been the means of giving him many friends among those whom he had only known as acquaintances before. He was convinced that the step taken by the Society was a hopeful one and a right one. The difficulties had been great, but these difficulties were now practically at an end. There were many ways in which they could increase their funds if it were necessary. If the lease of the Berner Street premises should come to an end before the Society had paid off a corresponding amount of the debentures, he was certain that they could easily find the necessary money to keep their finances in good

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condition; and was confident that next year they would be enabled to pay off more than the minimum amount stipulated in the deed. He congratulated them on the choice of his successor in the person of Dr. Church. He would carry away, on relinquishing his post, associations, and pleasurable souvenirs which would remain with him for the rest of his life; and concluded by reading a letter from the junior Treasurer, Mr. Bostock, explaining that ill health prevented his attendance.

Mr. CROFT moved, and Dr. CHAMPNEYS seconded—"That the best thanks of the Society be given to the retiring members of Council, Dr. Duffin, Dr. Pye-Smith, Dr. Eastes, Dr. Whipham, Mr. Davies-Colley, and Mr. Pearce Gould, for their services to the Society during their term of office."

This was carried by acclamation, and the vote was acknowledged by Dr. GEORGE EASTES on behalf of his absent colleagues, who remarked that he had had a good deal of experience of work on the councils of similar societies, and he was in a position to say that in no other society was there anything like so much work for the council as this one, owing to the important and multifarious business carried on.

The CHAIRMAN then called upon the Scrutineers for their report, and announced the result of the ballot to be as follows:

President.—Jonathan Hutchinson, F.R.S.

Vice-Presidents.—Charles John Hare, M.D.; Frederick William Pavy, M.D., LL.D., F.R.S.; J. Warrington Haward, Thomas Pickering Pick.

Honorary Treasurers.—William Selby Church, M.D.; John Ashton Bostock, C.B.

Honorary Secretaries.—John Mitchell Bruce, M.D.; Rickman J. Godlee, M.S., M.B.

Honorary Librarians.—Samuel Jones Gee, M.D.; John Whitaker Hulke, F.R.S.

Members of Council.—Sidney Coupland, M.D.; Frederick Thomas Roberts, M.D.; Frederick Taylor,

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M.D.; George Thin, M.D.; Samuel West, M.D.; William Henry Bennett; Alban Henry Griffiths Doran; Reginald Harrison; Charles Higgens; Thomas Fitz-Patrick, M.D.

The CHAIRMAN read a letter from the President elect, Mr. Jonathan Hutchinson, expressing regret at his inability to be present, and proposed that the new President be invested with the badge and presented with the President's master-key at the next ordinary meeting of the Society.

A vote of thanks to the Scrutineers for their services terminated the proceedings.

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Receipts.

	£ s. d.	£ s. d.	£ s. d.
To Balance on 1st January, 1893 :			
Cash in hand		92 8 7	
,, at Bankers		623 5 10	
			715 14 5
,, *Subscriptions, Fees, &c.* :			
431 Annual Subscriptions at £3 3*s.* ..	1357 13 0		
22 Entrance Fees at £6 6*s.* ..	138 12 0		
9 Non-resident Subscriptions at £1 1*s.*	9 9 0		
Fine	1 0		
		1505 15 0	
,, *Transactions* and *Proceedings* :			
Sold by Messrs. Longmans (*Transactions*)	53 2 11		
,, Librarian (*Proceedings*) ..	10 1 0		
,, ,, (Catalogues) ..	15 0		
		63 18 11	
,, *Rents received*		2461 16 6	
,, *Interest* :			
On Permanent Endowment Fund ..	12 14 0		
From Prudential Assurance Co. ..	8 9 8		
		21 3 8	
			4052 14 1

CHARLES J. HARE,
J. A. BOSTOCK, } *Treasurers.*

£4768 8 6

MARSHALL HALL

(TRUSTEES: *Walter Butler Cheadle, M.D.*,

The amount of Stock (Consols) standing to credit of this

CHARLES J. HARE,
J. A. BOSTOCK, } *Treasurers.*

PERMANENT

(TRUSTEES: *Sir Andrew Clark, Bart., M.D. (deceased)*,

New South Wales 4 % Inscribed Stock

CHARLES J. HARE,
J. A. BOSTOCK, } *Treasurers.*

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Payments.

	£ s. d.	£ s. d.
By *Rent, Rates, and Taxes*		224 12 10
„ *Lighting (for five quarters), Heating, and Cleaning*		385 13 7
„ *Repairs, Furniture, &c.*		19 15 7
„ *Meeting Expenses*		11 13 0
„ *Printing, Stationery, and Stamped Envelopes*	178 9 1	
Stamps (other than the above)	7 0 0	
		185 9 1
„ *Officers and Servants:*		
Salaries and Wages		864 17 7
„ *Library*:—Books and Binding		229 17 3
„ '*Transactions*' and '*Proceedings*'		460 8 11
„ *Extraordinary Charges:*		
Science Committee—("Spas")		15 5 8
„ *Debentures*, Interest on		1427 15 0
„ *Annuity*		8 14 0
„ *Auditors' Fee*		10 10 0
„ *Bank Charges* and Cheques		5 13 4
„ *Miscellaneous Payments*		33 3 5
		3883 9 3
„ *Payments on Account of Building, &c.:*		
Rent-producing Alterations (W. Robertson)—		
Smaller North Room £80 19 5		
Staircase Room 39 0 7		
	120 0 0	
Former unsettled Accounts and Contracts:		
Building (B. Nightingale)	50 0 0	
Building and Heating Apparatus (Johnstone, Norman, and Co.)	60 0 0	
Accountants' and Auditors' Charges	59 16 9	
	289 16 9	
Less Proceeds of Sale of Electric Plant	180 11 6	
		109 5 3
		3992 14 6
Balance:		
Cash in hand	135 10 10	
At Bankers	640 3 2	
		775 14 0
		£4768 8 6

MEMORIAL PRIZE FUND.

William Ogle, M.D., and Mr. Thomas Smith.)

Fund on the 31st December, 1893, was ... £596 5 2

ENDOWMENT FUND.

Walter Butler Cheadle, M.D., and Mr. Alfred Willett.)

.. .. £326 7 3

Audited and approved:

WOODBURN KIRBY, MUNDY, & Co.,
Chartered Accountants,
19, Birchin Lane, E.C.

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LIST OF PAPERS.

N.B.—The Council of the Royal Medical and Chirurgical Society deem it proper to state that the Society does not hold itself in any way responsible for the statements, reasonings, or opinions set forth in the various papers which, on grounds of general merit, are thought worthy of being published in its 'Transactions.'

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ADDRESS

OF

WILLIAM SELBY CHURCH, M.D.,

SENIOR VICE-PRESIDENT,

AT THE

ANNUAL MEETING, MARCH 1ST, 1894.

THE Society has met to-day under unprecedented circumstances. For the first time in its history the Presidential Chair is vacant. The illness and lamented death of our President at the very commencement of the present winter session has thrown a shadow over what otherwise would have been a most successful year, and deprived the Society of the services of one who, whilst he filled the Chair, devoted himself to furthering the best interests of the Society in a way which could not be surpassed and has seldom been equalled. To use his own words, which I heard him make use of on more than one occasion, "The College of Physicians has the first claim on my time, the Royal Medical and Chirurgical Society the next." How fully Sir Andrew Clark acted up to these words is best known to your Council, from whose deliberations he was rarely absent; yet that statement gives but an imperfect idea of the amount of time and thought that he bestowed on the Society and its welfare.

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You have already heard in the Report of your Council the steps which were taken upon the death of the President, and how it happens that I have the honour of addressing you to-day.

Those who were present at the last Annual Meeting, or who have read the address the President then delivered in the last volume of our 'Transactions,' will remember that in it he reverted once again to a subject which has at intervals been brought before the Society—the organisation of a Royal Academy of Medicine out of the existing Societies; and he concluded by saying, "I hope at no distant date to have the opportunity of suggesting for serious consideration the outlines of a plan for the creation by federation of a Royal Academy of Medicine and Surgery." If he had been spared to address you to-day, I think we should have heard from him a further development of his views on this most important proposal; for during last autumn, very shortly before his return to town, Sir Andrew Clark had a long conversation with me on this subject, and it is very greatly to be regretted that by his untimely death any plan for carrying out this most desirable scheme should have lost the benefit of his great influence, clear judgment, and powers of organisation.

Besides our President we have lost three of our Honorary Fellows and eleven Fellows. Though this number is not perhaps above the average, it is seldom that the mortality has been so heavy among the younger Fellows; and we have to deplore not only those who have added lustre to our Society, but several who, had they been spared, would without doubt have held foremost places among their fellows. I have, in accordance with the custom of the Society, prepared short obituary notices of them all; but I must say that I think, with one of your former Presidents, that it is very doubtful if this custom should be continued. If there is any value in these notices, they should be accurate accounts, vouched for by your President, of the professional lives of the

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Fellows, for reference in future years, when it might be difficult to obtain authentic information. To obtain these details necessitates a large amount of labour for your Presidents, and it is impossible that they can be put together in such a way as to be made interesting; furthermore, obituary memoirs have already appeared in the medical, and often in the daily journals. To-day, therefore, with your permission I shall only read portions of what I have prepared.

The first on my list should have properly been treated of last year, but being a non-resident Fellow, his death escaped notice until after the Annual Address had been delivered.

George Winter Rhodes was born at Huddersfield December 5th, 1822. He received his early education at the West Riding Preparatory School, Wakefield, and at the Temple School, Brighton. At the latter place he met with an accident to one of his feet, which ended his school days, and debarred him from active exercise for some years. In 1840 he was apprenticed at the Huddersfield Infirmary, and in 1843 entered King's College Hospital. He passed the Membership Examination of the Royal College of Surgeons in 1846, and the Licentiateship Examination of the Apothecaries' Company in 1847. In the same year he married the daughter of J. P. Peacock, Esq., and went to Paris, where he studied for some time, and on his return, after practising in London for a short time, settled about the year 1849 or 1850 in Huddersfield. In 1862 he was elected Honorary Surgeon to the Huddersfield Infirmary, and Medical Officer to the Workhouse; he also was appointed in the same year Medical Officer of the North District and Public Vaccinator of the Central District of the Huddersfield Union.

Mr. Rhodes took an active part in the literary and educational work of his native town. He was the first President of the Huddersfield Literary and Scientific Society, in connection with which body he acted as Honorary Local Secretary for the Government Science

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and Art Department. After several months of failing health more serious symptoms set in, leading to his death on the 22nd of November, 1892, within a few days of his seventieth birthday.

Septimus William Sibley was born in Great Ormond Street in 1831; his father held the responsible position of architect and surveyor to the county of Middlesex. Mr. Sibley received his early education at University College School, where he very greatly distinguished himself by his proficiency in mathematics; Mr. Edward Routh, afterwards Senior Wrangler at Cambridge, and he were rivals for the chief mathematical prizes in 1845 and 1846, the former obtaining first place in the subject of pure mathematics, and Mr. Sibley beating him in applied mathematics and experimental physics.

Endowed with great natural ability, and well trained by his mathematical studies, his successes were scarcely less brilliant when he turned his attention to medicine. Entering as a student at University College Hospital, and, as the custom then was, at the Middlesex also, he obtained the Gold Medal in Medicine and the Silver one in Surgery in the year 1851, and qualified as a Member of the Royal College of Surgeons in the succeeding year. Five years later he took the Fellowship of the College. His first hospital appointment was that of Apothecary's Assistant at the Middlesex Hospital in 1851, and he remained closely associated with the Middlesex Hospital for the rest of his life. In 1853 he was appointed Registrar to the Hospital, a post which he held until 1860. On the resignation of Mr. Mitchell Henry in 1856, Mr. Sibley became joint Lecturer with Mr. Nunn on Morbid Anatomy; this lectureship he held first in conjunction with Mr. Nunn, then with Dr. Murchison, and finally with Dr. Cayley until the year 1869.

In 1856 Mr. Sibley entered into partnership with Mr. Thomas Farquhar Chilvers, and from that time until his retirement from active practice may be said to have held the foremost place among the general practitioners

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in London. The high estimation in which he was held is evidenced by the fact of his being the first general practitioner elected on to the Council of the Royal College of Surgeons, where for many years he represented that branch of our profession. Mr. Sibley was a prominent member of the British Medical Association, a member of its Council from 1881 to 1891, Vice-President of the Parliamentary Bills Committee, and a member of many other committees of the Association; he was for many years the Treasurer of the Metropolitan Counties Branch, and filled the office of President of that Branch of the Association in 1878.

Notwithstanding the incessant cares of a large and successful practice, Mr. Sibley devoted a large amount of energy to the furtherance of all movements calculated to improve medical education or to assist in promoting the welfare of his fellow-men, and hence it was that he freely gave up much of his time to work in connection with charitable and scientific bodies. He was, until a year or two before his death, a member of the Middlesex Hospital Medical Committee; for ten years he acted as Chairman of the Dental Hospital of London; he was on the Executive Committee and for some years Treasurer of the Medical, Sickness, Annuity and Life, Assurance Society; Vice-President of the Royal British Nursing Association, and of the New Sydenham Society.

Such leisure as his busy life allowed him he mainly occupied in scientific pursuits; a keen observer of the habits and forms of animal and vegetable life, and a collector of natural history objects, he became an authority on many subjects quite outside his profession. To medical literature he contributed but little: his principal papers were four communicated to this Society; they are noteworthy for the care and completeness with which they are written.

In the summer of 1891 Mr. Sibley first showed symptoms of failing health, and was persuaded by his friends and family to take a holiday. Some weeks spent at

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Carlsbad appeared to benefit him, and he returned to his usual work and occupations until the following summer, when he was seized with right-sided hemiplegia and aphasia. From this paralytic attack he never made a satisfactory recovery, although he recovered sufficiently to be able to walk with a stick round his country property at Bletchingley, where he spent the last few months of his life. On the 15th of March further hæmorrhage in the brain took place, terminating fatally in the course of a few hours.

His courteous manners, and warm, generous and sympathetic nature endeared him to all who knew him, and his memory will long remain among us as an example of an accomplished and highly honoured practitioner.

Mr. Charles Edward Henry Cotes died at Bournemouth on May 4th, at the early age of thirty-three. He was the younger son of the late Major Cotes, R.A., and was educated at St. Paul's School and Caius College, Cambridge, where he took his B.A. degree in 1882. Mr. Cotes had entered as a student at St. George's Hospital before he went to the University, and after graduating at Cambridge returned to St. George's Hospital and continued his medical studies there, and became a Member of the Royal College of Surgeons in 1883, obtaining the B.M. and M.C. degree at Cambridge in 1884, and the Fellowship of the Royal College of Surgeons in 1885. In St. George's Medical School Mr. Cotes' abilities and aptitude for teaching were quickly recognised. After filling the post of House Surgeon in 1885 he was appointed Anæsthetist in 1887, Surgical Registrar in 1889, and Demonstrator of Anatomy in 1891; ill-health prevented his discharging the duties of this last post, but as Surgical Registrar his classes were highly appreciated and numerously attended. In addition to the posts he held in the Medical School at St. George's, he became in 1888 Surgeon to the Great Northern Hospital and to the Wells Street Branch of the Seamen's Hospital, and in

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the following year he was elected Surgeon to Out-patients at the Lock Hospital.

Vigorous in body as well as mind, Mr. Cotes distinguished himself whilst at Cambridge both on the river and in the football field. His simple, frank, and unaffected nature rendered him then, as afterwards, deservedly popular with all with whom he was brought in contact. Teaching was with him a pleasure, and the hours spent in his company are looked back to by his pupils with affectionate regret. The zeal with which Mr. Cotes pursued his professional work led him to overtask his strength, and in 1890 symptoms of pulmonary mischief led to his resigning some of his hospital appointments. In 1891 he had a serious attack of pneumonia, but was so greatly benefited by a voyage to Australia that he hoped that all serious disease was arrested. This, alas! was not the case, as in the autumn of 1892 his health again broke down; unmistakable signs of phthisis declared themselves, and led to his premature decease.

Mr. Cotes was a member of the Clinical Society, to whose 'Transactions' he contributed two papers, and of the Medical Society. His connection with the Lock Hospital afforded him opportunities for writing a valuable paper on the "Treatment of Acute Gonorrhœa," in which he advocated a novel mode of treatment, and supported his views by a large number of successful cases.

Marcus Beck was born in the year 1843 at Isleworth, where his father had settled down to a commercial life after having spent his early years in the mercantile marine service. His mother was a Miss Lucas of Hitchin, belonging to a family well known and much respected in Hertfordshire; through his mother he was connected with the family of the celebrated Dr. Thomas Young, and on his father's side with the Listers, his paternal grandmother being the sister of Mr. Joseph Lister, the inventor of the achromatic microscope and the father of the present Sir Joseph Lister. He thus was descended on

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both sides from families which had distinguished themselves in science, and he shared in their hereditary ability.

Marcus Beck received his early education at Queenwood College under Mr. G. Edmondson, and subsequently was under the tuition of Mr. Arthur Abbott at Hitchin. In 1860 he commenced his medical education at Glasgow, where his relative Sir Joseph Lister was then Regius Professor of Surgery. In 1863 he left Glasgow and entered as a student at Sir Joseph's old School, University College Hospital, where he very quickly gave proof of unusual ability. He passed the Membership of the Royal College of Surgeons in 1865, and served as House Surgeon to Mr. Erichsen (1866), and Physician's Assistant to Dr. Hare (1867). In 1867 he graduated as M.B. at the London University, obtaining the University Scholarship in Medicine, and the following year gained the gold medal in Surgery, and took the M.S. degree. In 1869 he became a Fellow of the Royal College of Surgeons. At University College Hospital, besides filling the posts of House Surgeon and Physician's Assistant, he successively held the posts of Demonstrator of Anatomy under Professor Viner Ellis, Surgical Registrar (1870), Assistant Surgeon (1872), Teacher of Operative Surgery (1875), Professor of Clinical Surgery (1883), and in 1885 succeeded to the late Professor Marshall in the Chair of Surgery. In 1890 he was elected on to the Council of the Royal College of Surgeons, and became a member of the Court of Examiners.

Coming of an old Quaker stock, Beck possessed the virtues which so often distinguish it—a strong sense of duty, honesty of purpose and thoroughness in all work undertaken. The commencement of his scientific work was the elaborate analysis of the surgical cases in the practice of the hospital, which were first issued when he became Surgical Registrar. Though his original work was not large it was of very high quality. The amount of labour and careful research which he devoted to the

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compilation of the Catalogue illustrative of the Specimens of Surgical Pathology in the Museum of University College can hardly be calculated. Not less laborious or valuable was the part that he took in investigating the nature and causes of pyæmia, septicæmia, and purulent infection when serving on the committee of the Pathological Society appointed for this purpose in 1879. His article on "Diseases of the Kidney Secondary to Affections of the Lower Urinary Tract" is perhaps his most important contribution to medical literature; but he also wrote valuable articles on "Surgical Kidney," "Erysipelas," and some other subjects for Quain's 'Dictionary of Medicine,' and ones on "Diseases of the Breast and Perityphlitic Abscess" in Heath's 'Dictionary of Surgery.' He edited the eighth and ninth editions of Prof. Erichsen's 'Science and Art of Surgery' with singular skill and judgment, bringing the work up to date whilst preserving the characteristic style of the original author.

As a teacher Beck was conspicuously successful; his success was not a little due to the extreme pains he took in preparing himself for this duty, which was with him a duty of love. One who knew him most intimately says, "Perhaps no one but myself knows the enormous amount of work which he did with the sole object of being able to teach the students thoroughly. The study of his own cases, and the research which he carried out in connection with them, were both as exhaustive as possible. His teaching was thoroughly appreciated, and his clinical and systematic lectures were always thronged. He threw into his hospital work an amount of patience and zeal which few but he have the physical strength to give; and it is remarkable that in spite of his diabetes his physical strength was enormous, and continued so up to eighteen months before his death."

As a practical surgeon Beck was a skilful operator, and was an early exponent of the advantages of antiseptic means in surgery. With all his excellence as a surgeon, and associated with Professor Erichsen for many years as

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his private assistant, it is somewhat remarkable that Beck had not a larger private practice. This was partly due to his heart being in his hospital and teaching work, and partly to his health. Aware for many years of his tendency to the disease which eventually proved fatal, he withdrew himself in a great measure from London life and society.

As a young man Beck was remarkably handsome and of great bodily vigour, delighting in outdoor exercise, which until the last year or two of his life always had a most beneficial influence on his health; and the first serious symptom of failing strength was the fact of his not gaining weight during his summer holiday. He was colour-blind to red, a peculiarity which it is believed his illustrious relative Dr. Thomas Young also shared in.

It would be impossible to speak too highly of Mr. Beck's personal character; beneath an occasionally somewhat cynical manner lay a truly noble nature, and a genuine kindness of heart which attracted all who came into relationship with him. Originally a member of the Society of Friends, he withdrew from their body, but never formally joined any other religious denomination. His final illness was short; attacked by influenza on the 14th of May, he appeared by the 20th to have passed through all danger, when cerebral symptoms set in and were succeeded by coma, in which he quietly passed away on the 21st of May.

At the last meeting of the Clinical Society for the Session 1892–3, those present heard with the deepest regret the President announce the sad news of the death of its senior Secretary, *Dr. Walter Baugh Hadden*, which had occurred on the morning of the day of meeting, May 26th. Dr. Hadden fell a victim to an attack of pneumonia, which was not improbably septic in nature, and caused by an accidental wound when making a post-mortem examination. He may therefore be said to have given his life, as he had given his life's work, to the cause of medical science. From early boyhood—for he commenced his work as a medical student at the unusually

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early age of sixteen—he laboured with exemplary diligence and corresponding success in the profession he had chosen.

After passing with the greatest credit to himself through the usual routine of a student's career, and after filling at St. Thomas's Hospital various posts in the Medical School, he was just about to exchange the post of Senior Assistant Physician for that of Physician to the Hospital, when his premature death cut short a career which, highly honorable as it was, gave promise of a still more distinguished future.

Although Dr. Hadden was not a contributor to our 'Transactions,' he left behind him much excellent work, distinguished by its originality and usefulness. As Honorary Secretary to the Clinical Society's Committee on Myxœdema, he took a share in the very laborious investigations into that malady which are embodied in its Report, and wrote independently two papers on that disease, one of which appeared in 'Le Progrès médical,' the other in 'Brain.' Soon after taking his Doctor's degree at the London University he went to Paris, and placed himself under the guidance of Professor Charcot; his intimacy with the latter's teaching and mode of study led to the Sydenham Society placing in Dr. Hadden's hands for translation Charcot's 'Lectures on the Localisation of Cerebral and Spinal Diseases,' and it was probably the influence of his old master which caused him to direct his attention mainly to nervous diseases. Dr. Hadden's connection with the Children's Hospital gave him facilities for the observations he made such happy use of in his papers on "Head-nodding" and "Head-jerking in Children," and on "Certain Defects of Articulation in Children." Numerous other articles in the 'Dictionary of Medicine' and periodical medical literature still further attest his industry and ability.

Dr. Hadden was the youngest son of Mr. Robert Hadden, one of the proprietors of the 'Liverpool Courier;' he was born in 1856, and commenced his medical studies at the Royal Infirmary, Liverpool, when quite a boy, in

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1873. Three years later he entered at St. Thomas's Hospital, and passed his examination for the Membership of the Royal College of Surgeons in 1877 as soon as he was of age. Dr. Hadden's talents and industry were quickly recognised at St. Thomas's Hospital, where he was appointed (1879) Medical Registrar the same year that he became M.D. of London University; this post he held with two years' interval until 1887. In 1882 he became Demonstrator of Morbid Anatomy. In 1887 he was elected Assistant Physician to the Hospital, Lecturer on Materia Medica in 1890, and Joint Lecturer on Pathology in 1891. Besides the posts that he held at St. Thomas's Hospital he was also Assistant Physician to the Great Ormond Street Hospital for Children, and Physician to the Royal Hospital for Women and Children, Waterloo Road; he was also an Examiner in Materia Medica at the Royal College of Physicians, of which body he became a Fellow in 1888.

Charles Derby Waite was born in 1807 in the same house in which he lived the greatest part of his life, and died, 3, Old Burlington Street, where his father had been settled in practice as a dentist, and in that capacity had been connected with King George the Fourth. Mr. Waite was educated at the Charterhouse, and at the early age of sixteen was apprenticed to Sir Charles Bell, and entered on his medical studies at the Middlesex Hospital. He became a Member of the Royal College of Surgeons in 1829, and in the same year filled the post of House Surgeon at the Middlesex Hospital. He subsequently studied in Paris, and in 1834 he entered as a pensioner at Peterhouse, Cambridge, and kept the nine terms then requisite for the Bachelor of Medicine degree. Mr. Waite, however, did not proceed to his M.B. degree until the year 1847. Previously to his stay at Cambridge Mr. Waite had been attached to the Commission of Inquiry into the health of the Metropolis during the prevalence of Asiatic cholera in 1832.

Mr. Waite was in easy circumstances, and does not

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appear to have at any time engaged much in private practice. At one period of his life he was offered and accepted the post of resident physician in the family of the Earl of Warwick during the illness of the Countess in 1851–2, and filled a similar position from the years 1854 to 1860 to Lady Kerrison, the widow of Sir Edward Kerrison, K.C.B., a distinguished Peninsula and Waterloo officer. For many years also Mr. Waite acted as Honorary Physician to the Westminster General Dispensary, which old-established charity highly appreciated his services, and for a long period he was Consulting Physician to the English and Scottish Law Life Assurance Society.

Dr. Waite died unmarried on June 13th, and will be affectionately remembered by those who knew him for his many amiable qualities and kindness of heart.

Jean Martin Charcot, elected an Honorary Fellow in 1883, was born in Paris, November 29th, 1825. His father was a coach-builder, and not in a large way of business. His father's means did not allow of his giving all his three sons a liberal education, and he is reported to have said to them whilst still mere boys, "I am not able to give you all a classical education: whichever of you shall have done best at the end of the scholastic year shall continue his studies, another shall be a soldier, and the third a coach-builder." As the result of the year's work Jean Martin was sent to the Lycée Saint-Louis, and in due course entered on his medical studies. He became an "Interne des Hôpitaux" at the Salpêtrière in 1848, passed the doctorate in 1853, and for the next two years filled the office of "Chef de Clinique Médicale." He used in after years to record with pride that during this early portion of his career he was enabled, by giving instruction to private pupils, to repay to his family the greater part of the pecuniary sacrifices they had made for his education. In 1856 he was appointed Physician to the Central Bureau of Paris, and four years later Professor Agrégé in the University.

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It is interesting to note that he chose for the subjects of his theses for his examinations those which were destined to bear much fruit in after years. "Chronic articular rheumatism, nodosities of the joints, and asthenic gout" was the subject of his thesis for his doctor's degree; whilst for his other examinations he chose "The expectant treatment in medicine" and "Chronic pneumonia."

In 1862 Charcot returned as Physician to the Salpêtrière, and never left it again. Under his direction this hospital, which had been little more than an asylum for the aged and infirm, became the centre for the accurate clinical study of the most difficult problems that can come before us.

In 1866 he commenced to give at the Salpêtrière those lectures with which his name will be ever associated, and which have had so great an influence on our views of nervous diseases. At that time the theatre, which many of you know, was not built, and Charcot made use of any room which happened to be vacant. His lectures quickly attracted attention, and from this period to the end of his life Charcot probably exercised the greatest influence of any modern teacher in medicine, students both old and young flocking to his clinique from all parts of the civilised world.

In 1872 he was appointed to the Chair of Pathological Anatomy in the University of Paris, succeeding his friend Mons. Vulpian in this Chair, one of the highest honours that the University of Paris on its medical side has to bestow. During the ten years that Charcot held the Professorship of Pathological Anatomy he continued his lectures at the Salpêtrière, dealing chiefly with locomotor ataxia, spinal amyotrophia, post-hemiplegic hemichorea, and tabes dorsalis; he took, moreover, an active part in assisting and contributing to medical periodical literature. In 1882 a special Chair of Clinical Neurology attached to the Salpêtrière was founded, and Charcot, who had virtually been performing the duties of this chair for sixteen years, was naturally its first occupant. He had previously, in 1873, been elected to the Membership of

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the Academy of Medicine, and in 1883 became a Member of the Institute of France.

I have thus briefly put before you the principal incidents in Professor Charcot's student and professional life, but doing so gives but an imperfect idea of the man. Charcot was by nature endowed with artistic genius (I believe that it was want of means in early life that prevented him from becoming an artist), and to this artistic faculty much of the charm of his writings is due, and the same gift rendered his lectures so attractive; their subject-matter, however intricate, was always discussed in language at once precise and expressive, whilst the salient features of the subject under consideration were brought out with graphic force and clearness. It is too much the custom now to look upon Charcot as a specialist in nervous diseases; he was that and much more, for we must not forget the breadth of the subjects included in his early lectures at the Salpêtrière. To his influence and example the general spread in France of the use of the thermometer for clinical purposes was not a little due; nor must we forget his lectures on the maladies of the lungs, the liver, and kidneys, or those on the pathogenic conditions of albuminuria. Whilst he held the Chair of Pathological Anatomy much valuable work on general as well as special pathology emanated from him and his pupils.

In recent years Professor Charcot's name has been prominently brought before the general public in connection with hypnotism, and the laity have the most erroneous ideas as to Charcot's work and writings on the subject. Hypnotism had more or less occupied his attention for years; his researches in 1876 into *métalloscopie* and *métallothérapie* naturally led on to further ones in hypnotism. In the former Charcot bears frequent witness to the value of Braid's original work. It would have been well if all inquirers into these subjects had studied them in the careful and cautious way that Charcot did; his long familiarity with the varying mani-

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festations of disorders of the nervous system enabled him to approach the subject with a mind not only free from scepticism as to the reality of the phenomena, but also especially trained to seize on the salient and important points. He has himself summed up the method that should be used in these investigations; instead of going in pursuit of the unexpected and extraordinary, it should be our endeavour to confine ourselves to those clinical symptoms and physiological phenomena which are most easily appreciated, to restrict ourselves to the examination of the simplest and most frequently recurring symptoms, and only afterwards to consider the more complicated and fugitive ones, which do not appear connected with any as yet known physiological facts. It is because these simple precautions have been so often neglected that the researches into hypnotism have as yet led to no practical results, and indeed may be said not to have received from our profession in this country the consideration which their importance deserves.

To attempt to review his work or the influence it may have on the future of medicine is beyond my power, and, in fact, it appears to me that the time has hardly yet come when we can appraise the value of it. It may, however, be safely said that, in addition to all we owe to him as a pathologist, it is mainly to him and his work that the recent enormous strides in our knowledge of the dependence of certain forms of disease on disordered conditions of the nervous system are due.

In person Charcot bore a strong resemblance to the Bonapartes, especially to Prince Jerome Napoleon. A calm and thoughtful face, joined with a certain amount of reserve of manner, occasionally led those imperfectly acquainted with him to consider him cold and unsympathetic; his intimates, and those who had the privilege of seeing him in private life, know how far this was from being the case.

Placed by a fortunate and happy marriage at an early period of his career in easy circumstances, Charcot was able

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to apply himself with untiring and unremitting devotion to scientific medicine, and at the same time cultivate those artistic tastes to which I have already alluded. His reputation among French painters as a critic of both ancient and contemporary art stood high; and his articles in the 'Nouvelle Iconographie de la Salpêtrière' are examples of the combination of scientist and artist. His sketch-book was his constant companion when on his travels, and his facility as a draughtsman added to the charm and instructiveness of his lectures. Music and general literature, too, had their attraction for him. Shakespeare was one of his favourite books, whilst in music he delighted especially in Glück, Beethoven, and Weber.

Thoroughly acquainted with our medical literature, and speaking English well, few foreign professors have been so generally known or had so great an influence in this country as Professor Charcot; he was an honorary member of nearly all our leading medical societies, and an honorary member of the British Medical Association, which body in 1890 paid him the unusual compliment of asking him to deliver the Address on Medicine at the Annual Meeting in Birmingham.

It was with great regret that the English medical world heard that, on account of failing strengh, he felt himself unable to accept the invitation; though the world knew it not, he was probably aware that his health was failing. Whilst lecturing one day last summer he fainted, and though not much alarm was felt at the time, his condition gave anxiety to his friends. Accompanied by his two friends MM. Debove and Strauss, he went for change and rest on an excursion to the shores of the little Lake of Settons in the Nièvre, and died quite unexpectedly of an attack of angina pectoris on August 16th, in the sixty-ninth year of his age.

By the death of *William Morse Graily Hewitt* on August 27th, one of the most distinguished teachers of gynæcology in our metropolitan schools was removed from us. For some years prior to his death it was obvious

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to those only slightly acquainted with him that his health was failing, whilst his more intimate friends were aware that for many years he had been struggling against serious disease, and his death, though at the last sudden, was not unexpected. He was born at Badbury, Wiltshire, in 1828, and received his early education at the Gloucester College School, coming to London as a resident pupil with Mr. H. Burford Norman in 1845; he entered as a medical student at University College Hospital, and matriculated at the London University the following year.

His University career was one of unbroken success, obtaining the Gold Medal in Chemistry in 1848, and graduating as M.B. in 1850 with honours in all four subjects. After taking his Bachelor's degree Graily Hewitt went to Paris, and attended chiefly the clinical teaching of Prof. Trousseau at the Hôpital des Enfans Malades. On his return to England he passed the examination for the Army Medical Service, but never joined the service, and in 1852 finally settled in London, becoming Surgical Registrar and Superintendent to St. Mary's Hospital, then freshly established. In the following year he married the only daughter of Mr. W. Hollis, of Northampton. On the opening of the Medical School of St. Mary's Hospital in 1854 he was appointed Lecturer on Zoology. In 1855 he took the degree of M.D. at the London University, and became the same year a Member of the Royal College of Physicians. By 1858 Dr. Graily Hewitt had apparently determined on his future line of practice, for he took in this year, along with the late Dr. Tyler Smith, an influential part in establishing the Obstetrical Society, of which he was the first Honorary Secretary, and for the remainder of his life a regular attendant on its meetings, and a most important and constant contributor to its 'Transactions.' After filling the office of Vice-President, he was in 1869 elected President of the Society.

In 1858 he became Physician to the Samaritan Hospital for Women, and two years later was appointed joint

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Lecturer with Dr. Tyler Smith on Midwifery and the Diseases of Women and Children at St. Mary's Medical School, and in 1863 Assistant Physician-Accoucheur to the hospital. At this period of his career Dr. Hewitt was closely associated in private practice with the late Dr. Rigby, and in 1861 he moved from Radnor Place to Dr. Rigby's house in Berkeley Square.

In 1865 he was elected to the Professorship of Midwifery in University College, and Physician-Accoucheur and Director of the Obstetrical Department to the hospital, a post he filled for twenty-one years, and was in 1866 elected a Fellow of the Royal College of Physicians.

Dr. Graily Hewitt was a prolific writer, and it would be profitless here to enumerate the papers which he contributed to the medical journals and periodicals; by no means the least important among them is the paper on Whooping-cough read before the Harveian Society in 1855, and subsequently published.

In later years his writings were almost entirely devoted to gynæcological subjects. His principal work, entitled "The Pathology, Diagnosis, and Treatment of Diseases of Women," was first published in 1863, and has gone through several subsequent editions; from the date of its appearance until his death Dr. Hewitt may be said to have been the chief exponent, in this country at all events, of what may be termed the mechanical system of uterine pathology. The value of his work in this direction must be left for future years to determine; to one who, like myself, is not fitted to judge of it, it appears that if the old adage, "meddlesome midwifery is bad," be true, meddlesome gynæcology is worse. Graily Hewitt himself wrote, "no one could be a good observer unless he was an active theorist;" and I think that in his writings, if not in his practice, he followed his maxim out to excess.

Though never robust, and during the later years of his life a frequent sufferer from serious illness, the amount of work that he got through was enormous. In addition to a large and fashionable practice he was a most diligent

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student of medical literature, and kept abreast of the most recent advances in science.

Dr. Graily Hewitt did not contribute any papers to our 'Transactions,' but he served the Society on its Council and Library Committee, and for two periods acted as a Referee. He was a Fellow of the Harveian, Clinical, and Pathological Societies, and an Honorary Fellow of many learned Societies, both British and foreign.

Increasing ill-health led to his retirement from active work in 1890, and though a winter spent in Egypt greatly mitigated the severity of his pulmonary troubles, his other ailments became intensified during last summer, and he quietly passed away, deservedly honoured by his profession and deeply regretted by his friends.

On November 6th the Society was deprived of its President, *Sir Andrew Clark*.

I feel sure that the Society will neither expect nor wish me to read them anything approaching to a full account of our late President's remarkable life; it is quite unnecessary to do so, as so many biographies of him have already appeared, not only in the professional journals, but in the public press. I shall, therefore, confine myself to supplementing these accounts with some details I have gathered from authentic sources of his earlier life, and to the correction of some errors which have accidentally crept in, and shall touch very briefly on some other portions of his career.

I will begin with his own account of his boyhood. On September 25th, 1885, he was entertained at a banquet in Dundee, of which a very full report appears in the Dundee 'Courier and Argus' for Saturday, September 26th. In responding to the toast of his health Sir Andrew said "he was afraid to say how many years ago he began life in Dundee; here it was that he got the first rudiments of a general education; here it was that he made his first acquaintance, and in some senses a happy acquaintance with the rudiments of medicine, under such men as Webster, Crichton, and Munro; here

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it was that he got that which had never until now failed him—the love of his work, which had sustained him always in doing it. Here, too, he had made acquaintances, some of which had ripened into friendships which had influenced his life; here, too, he had got the elements of religion, and those rules of conduct whereby he was to endeavour to guide himself along the dangerous and difficult highway of life." He then goes on to say, "towards the end of 1846 he left Dundee to enter upon the serious business of life;" and speaking of himself at that time, he says "he never saw his parents; he had very few friends, because he was always reserved in nature; he was in feeble health; he had no ambition; he was possessed of continually recurring sad moments; and he was sensible of the coming struggle for life, and indeed he had little expectation and not much desire to live." There can, I think, be no doubt that in these latter remarks Sir Andrew, with artistic effect, heightened the colours to contrast his earlier life with the position he subsequently attained, for I have been in communication with those who can give me their recollections of him, both in Dundee and Edinburgh, and they represent him in a very different light. Of his life in Dundee we get a glimpse in the speech with which the late Dr. Greig, the chairman at the banquet, introduced the toast of his health; in it he gives a vivid sketch of the medical life of Dundee, and of Sir Andrew's first teachers in medicine. "The Royal Infirmary was at that time in King Street, and the physicians and surgeons of the institution were men of no mean reputation in the healing art; and the mention of such names as John Crichton, Matthew Nimmo, Dr. Munro, and Dr. Webster will recall pleasant memories to many of us. I have no time to eulogise each separately, but will refer only to the last name I have mentioned, Dr. Webster. He was a shrewd, careful, observant physician and kindly man, much respected and liked by his fellow-practitioners and the general public. He devoted a good deal of his time

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to work in the Infirmary, and was amongst the first to point out the difference between typhus and typhoid fever, Dundee possessing at that time a most unenviable notoriety for several fever epidemics. Dr. Webster had his surgery up Albert Court in the Nethergate, and had one or two apprentices, one of whom was our guest of to-night. Who could have believed at that time that the surgery boy in Albert Court could have worked his way to the very top of his profession, and become the leading physician in London? However, there were prophets in those days, some who said the lad would come to something; and Sir Andrew will perhaps remember one old gentleman who said, 'You will go up to London, you will ride in your carriage, and you will be a knight yet.'"

Before his connection with Dr. Webster, Sir Andrew had been in a similar but still humbler position with Dr. Nimmo. I have been unable to ascertain when Sir Andrew first went to Dundee, but it was in quite early boyhood, and it was at the Tay Square Academy, and not at Aberdeen, that he obtained his early education, and at Dundee he began his medical studies. One who sat at the same desk with him at the Tay Square Academy, and spent many an hour with him in the small dingy room which was dignified by the title of surgery, writes thus of him:—"In person he had no decided marks at that early age of a delicate constitution, although I do not remember him taking part in our active youthful sports.

"He had a peculiar swing in his walk, which I think he retained in some degree to the last. Mannerisms, which in later life some were disposed to ascribe to affectation, were quite natural to him, and had grown up with him from boyhood. Even as a boy he was singularly pure and noble-minded, scorning everything that was mean. When he was not studying medicine he was writing moral essays, which he addressed as letters to a family of his acquaintance. These were often of considerable length, and his facility in composition, as well as his

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elevation in tone, at that early age were very remarkable. In conclusion I may say that I do not remember any of my old schoolfellows who were so earnest in study, or who set such a high ideal before them as did Andrew Clark; he seemed to live on a higher level than them all."

During the first portion of his life that I have been describing at Dundee, Sir Andrew Clark lived with a Mrs. McKenzie, who had acted as a foster-mother to him; she lived at 27, Wilkie Lane, a somewhat obscure locality. Sir Andrew looked upon Dundee as his home, for on his Diploma of Membership of the Royal College of Surgeons, England, granted on May 31st, 1844, beneath the line "Name and Residence of Member," is written in the same bold characteristic hand which he wrote through life "Andrew Clark, Dundee, Forfarshire."

I will pass on now from his Dundee life to Edinburgh, remarking, however, that in all the biographies that have appeared Sir Andrew Clark is stated to have studied at Aberdeen. I have shown that his boyhood was spent at Dundee, where, too, he obtained his early medical education, and I believe that from Dundee he went directly to Edinburgh. His old school companion, whom I have quoted above, says, "As far as I am aware he did not attend classes in the University of Aberdeen;" and on the same authority I can say that he was resident in Dundee till after the year 1839.

The Registrar of the University of Aberdeen has kindly searched the names of entrants in the junior class or first year for me, both at King's and Marischal Colleges, between the years 1833 and 1842, and the name does not occur; neither does the name occur in the class rolls during the same period, nor in the Medical Class lists of Marischal College between the years 1836 and 1844. It seems quite certain, therefore, that Sir Andrew was not a regular or curricular student in arts or medicine in Aberdeen; and where the entry of his name is found as taking the M.D. degree in 1854, he is described as M.R.C.S.Eng. and M.R.C.P.Lond., and no mention is made of his having either studied

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or graduated at Aberdeen. The Registrar also informs me that he has searched the bursary lists for King's College between the years 1832 and 1841, and the name is not there, and as it does not appear upon the class roll he thinks he could not have held one at Marischal College.

At Edinburgh Clark was an extra-academical student, and in the classes of the various extra-mural teachers carried all before him, receiving medals in almost all if not all the classes he attended.

It has been stated that he was a Demonstrator of Anatomy to Dr. Robert Knox; it may be so, but Knox's last course of lectures as a recognised teacher of anatomy in Edinburgh was delivered in the winter of 1841–2, and I am informed by one who was a pupil of Knox's at that time that Clark was not even a pupil of Knox's then, and certainly not a demonstrator. If Clark, therefore, was ever connected with Knox, it must have been after he ceased to teach anatomy in Edinburgh. Lonsdale, in his 'Sketch of the Life and Writings of Robert Knox,' says (p. 257), "Regardless of both legal and moral obligations, he [Knox] commenced lecturing on anatomy in Edinburgh in November, 1842, but got no class. He attempted a course of physiology in the following session with as little success." It is of course possible, but not probable, that Clark may have taken some part with Knox in these abortive attempts. Dr. Lockhart Robertson writes in the 'Lancet,' November 25th, that "in 1842–3 Clark was assistant to Dr. Hughes Bennett, then just appointed Pathologist to the Royal Infirmary, and used to demonstrate to students like myself, of his own age and standing, the morbid anatomy at the daily post-mortem examination." During the same period Dr. Robertson remembers him as attending the surgical lectures and clinical surgery of his father, Dr. John Argyll Robertson, and that the latter said to him, "Clark is the best student I ever had in my class; if you both live you will see him head of the medical profession."

It has been stated that whilst at Edinburgh Sir Andrew

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had typhus fever. This is an error. What really happened is rather amusing, and I venture to recount it. Towards the close of the year 1844 Professor Gairdner and Clark were both taken ill about the same time, and were thought by Dr. Alison to have typhus. This turned out to be the case with Professor Gairdner; but Clark, whose head had been promptly shaved, was out and about again in a few days wearing a velvet skull-cap, and only too glad to enjoy the laugh which his companions had at him. His career at Edinburgh and the date of his Diploma at the Royal College of Surgeons, England, May 31st, 1844, render it very difficult to accept the date of birth given in all the biographies, October 28th, 1826; for he would have been only sixteen when demonstrating under Hughes Bennett in the Pathological Department of the Royal Infirmary, and only seventeen and a half when admitted to the Membership of the Royal College of Surgeons, though then, as now, no member was supposed to be admitted under age. Shy and reserved in manner, and somewhat delicate in health, Clark made few intimate friendships at Edinburgh; but we see him gaining by his industry and ability the highest opinion of his teachers, and the respect of those few friends who did break through his reserve of manner.

After taking the Membership of the Royal College of Surgeons of England Clark returned to Edinburgh and continued his studies, especially those in pathology; it was during this period that he is best remembered by his Edinburgh acquaintances. He was a frequent debater at the Hunterian Medical Society, and even then was noticeable for the fluency and ornate character of his speeches. It was at this time that he wrote a series of lectures which have not been published, entitled "The Nature of some Recent Pathological Doctrines." Dr. Delépine,[1] in his very careful *résumé* of all Sir Andrew's papers, letters, and communications on medical subjects, says that these lectures were delivered at the Royal Infirmary,

[1] 'Journal of Pathology and Bacteriology,' vol. ii, No. 3, p. 265.

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but I have been unable to discover in what capacity he gave them.

Just such as he was at Edinburgh Professor Huxley describes him as being at Haslar, when he joined the naval service on September 1st, 1846. The Director-General of the Navy has kindly informed me that he was never employed afloat, being specially retained for service at Haslar, but not with the specific title of pathologist. At Haslar Clark's love of pathology and indefatigable industry caused him to institute microscopical demonstrations both pathological and clinical, and whilst there he delivered a series of lectures (in 1847) before the medical officers attached to the establishment, entitled "An Anticipation of the Views of Reinhardt on the Exudation Corpuscle," which are published in the 'London Medical Gazette,' and are the first published work of our late President. Serious symptoms of pulmonary mischief compelled him to obtain temporary leave of absence in 1847, and he took for his health a voyage to Madeira, but in 1848 he was at work again at Haslar.

During his life at Haslar Sir Andrew Clark matured, and threw away much of his reserve of manner, for, according to the description of a colleague of his there, "he was of a highly vivacious temperament, and showed the *perfervidum ingenium Scotorum* not only in the warmth of his arguments, but in the sensational and almost histrionic style of his abundant flow of speech. Outside pathology his favourite study was metaphysics, but for his age he was well read in many subjects." It was whilst at Haslar that he met and married his first wife, the only child of Captain John Forster, R.N., a member of the old Northumbrian family of Forster, who had followed Lord Cochrane and fought along with him in the Chilian War of Independence. After Mrs. Forster's death her daughter resided with an aunt at Gosport, and thus she was brought into the immediate neighbourhood of Sir Andrew; the marriage took place in 1851.

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Acting, I believe, on the advice of Sir John Richardson, Clark applied for the Curatorship of the Museum of the London Hospital, was appointed, and came to London in 1853.

I need not trace the rapidity with which Clark's reputation rose at the London Hospital; he gained the support of the leading members of the staff, and was elected, after a very close contest with Dr. Ramskill, Assistant Physician the following year. I will pass over the next fourteen years, in which his reputation both as a pathological and clinical teacher rapidly increased. During these years he lost his first wife, and four years later married again.

On August 14th, 1866, Sir Andrew became Physician to the London Hospital, and took a very active part in organising the arrangements for meeting the enormous amount of work thrown on the hospital by the epidemic of cholera which raged in the East End of London that autumn. It was in connection with this work that Sir Andrew first made the acquaintance of Mrs. Gladstone, and by permission I am allowed to read the following extract from one of her letters:

"The first time I had the privilege of meeting Sir Andrew Clark was in the London Hospital, during the outbreak of cholera in 1866. I was at that time a visitor in the wards, and circumstances thus led to my seeing a great deal of Dr. Clark, who, as Physician of the hospital, I constantly met.

"More and more I was struck, during that time of grave anxiety, by his ability, his zeal, his tender ministrations among the sick. The Convalescent Home at Woodford was the outcome of that cholera epidemic, there being thirty years ago a crying need for such an institution. I was deeply indebted to Dr. Clark for his invaluable aid in setting on foot the Woodford Home. He devoted much of his time and thought to it, and drew up the rules by which ever since it has been managed.

"I cannot better express the effect he produced on my mind than by saying I became convinced that my

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husband's health would be safe in his hands. Our son Herbert, then in his early youth, was, however, the first member of the family who came under his care; and we feel that, humanly speaking, his present excellent health is largely due to Sir Andrew's advice.

"From this beginning arose a long and most valued friendship. Not only was his professional devotion untiring, he was also a man whose strong religious character made him a rock to lean upon in all times of anxiety and strain.

"When I first introduced Dr. Clark to my husband as the physician I wished him to consult, my words were simply 'You would be safe in the hands of that great Dr. Clark.'"

For the next twenty years Sir Andrew continued his work at the London Hospital, becoming Consulting Physician in 1886. I need not allude here to the position which he gained during those twenty years, a position fitly marked in 1883 by his elevation to a baronetcy; but I do not think that the medical world at large fully appreciated Sir Andrew Clark's greatness, the breadth of his views, and the elevation of his character until after he had attained the high honour of being elected President of the Royal College of Physicians.

This is not the fitting place nor the time for me to dilate on the influence which in that position he exerted both on the profession and the general public, nor the amount of self-denying work which he cheerfully undertook in promoting the interests of both the science and art of medicine. No memoir, however short, should omit to mention his generosity and bounteous hospitality. Of his private generosity it does not become me to speak, but it was great. To this Society, and still more to the Royal College of Physicians, he was a generous donor; and I believe that a few years ago, had not the money been found from other sources, it was his intention to have given largely to the endowment of a Chair of Pathology in the University of Aberdeen.

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Sir Andrew Clark's life was one continuous and consistent whole. Knowing his own ability and confident in his powers, he determined to succeed, and his career is a noble example of what industry and ability can do. I have shown him to you as a boy at Dundee, without the advantages of either friends or education, determined to educate himself, and so impressing his companions and first masters in medicine with his powers that they foretold his future success. I have shown him to you at Edinburgh, carrying all before him in the extra-academic classes, and winning from one of his teachers the praise of "the best pupil I ever had." At Haslar we see him, notwithstanding his feeble health at that period of his life, beginning to take his place as a teacher, and lecturing to the most distinguished of his brother officers there. The same indomitable energy which enabled him to fight against his ill-health at Haslar was characteristic of him when he came to London, and had its fitting reward. His force of character, combined with the genuine sympathy he had for all in trouble or distress, caused him to gain the perfect confidence of those who consulted him, and was the secret of his remarkable professional success; whilst the elevation of his thoughts and his intellectual activity rightly enabled him to take his place not merely as the physician, but as the friend and companion of the leaders in science, of the poets, and of the statesmen of his time.

Dr. William Rhys Williams belonged to a medical family, his father being a physician of repute at Nottingham who, like his son, took great interest in mental disease, and was Visiting Physician to the Nottingham Asylum. Dr. Williams was educated at Merchant Taylors' School, where he obtained a classical scholarship and with it an entrance to St. Thomas's Hospital.

After qualifying as M.R.C.S. in 1858, he for a short time acted as assistant to a general practitioner at Daventry, and subsequently became Assistant Medical Officer to the Derby County Asylum, and from that time

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until his retirement from active work gave himself up to the study of insanity. After leaving the Derby County Asylum he for a time assisted Mr. Willett in the management of his private asylum, and then took the post of Assistant Medical Officer in the Three Counties Asylum, at that time under the supervision of the late Dr. Denne.

In 1866 he became Assistant Physician at Bethlem under Dr. Helps, whose health at that time was failing, so that almost the entire medical charge of the hospital fell on him ; and so well did he perform his duties that he was appointed Superintendent of Bethlem three years later, at the early age of thirty-two. For twelve years he held this post, which gave him scope for showing his ability in asylum management. During part of the time that he was Superintendent of Bethlem he lectured on mental disease at St. Thomas's Hospital. The success which had marked his management at Bethlem attracted the attention of the Government, and in 1878 he was appointed a Commissioner in Lunacy.

In 1862 he became a M.D. of the University of St. Andrews, and in 1869 joined the Pathological Society. He contributed a paper to the 'St. Thomas's Hospital Reports' on "Sudden Recovery in Cases of Insanity," and several shorter communications to the medical journals. Failing health necessitated his retirement from the post of Commissioner in Lunacy in 1889, and an attack of bronchitis terminated his life on November 28th, at the comparatively early age of fifty-six. He died at his residence at Leamington.

Dr. William Wegg died on December 3rd. He was one of our oldest Fellows, having joined the Society in 1844. I have been unable to obtain any information as to Dr. Wegg's family and preliminary education. He was a member of Gonville and Caius College, Cambridge, where he took the Bachelor of Medicine degree in 1843, proceeding to that of Doctor in 1848. He attended the practice of St. Thomas's Hospital, and dedicated the only

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work he published, 'Observations on the Science and Art of Medicine' (1851), to his old teacher, Dr. Henry Shuckburgh Roots. He became a Licentiate of the Royal College of Physicians in 1843, and a Fellow eight years later. Possessed of ample private means, Dr. Wegg appears to have long ago given up private practice. He was for some years Physician to the St. George's and St. James's Dispensary, and took an active interest in the welfare of the Royal College of Physicians, serving for two periods as Councillor, as Censor in 1867–8, and for upwards of thirty-five years acted as one of the Curators of the Museum. When first appointed Curator he expended much time and labour on the collection, and for the rest of his life took a lively interest in it and in the other business of the College.

The Society, too, benefited from Dr. Wegg's leisure, for in several capacities he served us faithfully and well. For four years (1854—1858) he acted as Honorary Librarian, having previously served on the Library Committee; he was a member of our Council in 1861–2, and held the office of Treasurer for seven years, from 1873 to 1880. Dr. Wegg did not contribute to our 'Transactions,' his only published contribution to medicine being the work alluded to above; but I believe that up to the time of his death he continued to take a lively interest in medical and scientific subjects, and was engaged in the preparation of a work on 'Philosophy and Medicine.'

John Tyndall, elected an Honorary Fellow in 1868, was born August 21st, 1820, at Leighlin Bridge, a village about six miles south of Carlow, but he claimed to be of English blood, his ancestors having nearly two hundred years previously emigrated from Gloucestershire, and were supposed to be descended from William Tindal, the translator of the Bible.

Tyndall's parents, though poor, filled a respectable position, and were able to give him a fairly good education, and for many years Tyndall maintained friendly relations and correspondence with his old schoolmaster Mr. John

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Conwill. In 1839, when the Ordnance Survey of Ireland was being made, Tyndall obtained a situation as "civil assistant" to the staff of engineers engaged on the work in his own county, and remained in that capacity for five years, having at that period of his life the intention of becoming a civil engineer. In 1844 he obtained employment with a firm in Manchester engaged in engineering work in connection with the railways which were then being projected in all directions. After spending in this manner some three years of very hard work, an intense desire to make himself acquainted with experimental science, and especially chemistry, led him to seek and obtain the post of teacher of mathematics and surveying at Queenwood College, then under the direction of George Edmondson, the Quaker, to whom is due the credit of first introducing the teaching of practical science into schools. It was at Queenwood that he formed his life-long friendship with Professor Frankland, who at that time was engaged in teaching chemistry there. The two mutually assisted each other, Tyndall receiving help and instruction in chemistry from Frankland, and the latter assistance in mathematics from Tyndall.

In 1848 these two young men, destined to become famous and to play important parts in the progress of scientific knowledge, left Queenwood, and went together to Marburg, in Hesse Cassel, to study science under Bunsen, to whom Tyndall in after years used to say that he owed obligations never to be forgotten. Here also he studied mathematics and physics under Professors Gerling and Knoblauch.

About the close of the year 1850 Tyndall left Marburg for Berlin, working there in the laboratory of Professor Magnus. Whilst at Berlin Tyndall attracted the notice of Dr. Bence Jones, and it is a satisfaction to us to remember that one of our own Fellows took a very considerable share in introducing Tyndall to the scientific world in London. Tyndall was made a Fellow of the Royal Society in 1852, and in the following year, not a little

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through Dr. Bence Jones's influence, he was invited to give a Friday evening lecture at the Royal Institution. The subject-matter of this his first lecture, delivered on February 11th, 1853, was "On the influence of material aggregation by the manifestations of force."—not, one would think, a very attractive subject for a Royal Institution audience but Professor Huxley tells us that "he captured his audience so completely that his appointment to the Fullerian Professoriate of Physics with the use of a laboratory, such as he needed for the original work that he loved, soon followed."

From the time of Tyndall's appointment as Professor of Physics at the Royal Institution until his death he occupied a foremost place among English physicists, and it would be difficult to overrate the influence he exercised in rendering the study of science more general in our present systems of education.

Just previous to his appointment at the Royal Institution he had stood for the Chair of Physics in Toronto, and, singularly enough, his intimate and constant friend Professor Huxley stood at the same time for the Chair of Natural History there. Fortunately for them and for this country, neither of these great men, destined to become leaders in their respective branches of science, were elected.

On Tyndall descended the mantle of his still greater predecessor Faraday, and as an exponent of science to the general public Tyndall was unrivalled. All who have ever had the pleasure of hearing him lecture could not fail to be struck by the interest with which the subject in his hands became invested, and by the charm and lucidity of the language in which he expounded it.

This is not the occasion nor am I qualified to express any opinion of the value of Professor Tyndall's work ; to those of us who can make no claim to special knowledge of physics the charm of his writings and the clearness of his descriptions left an impression, second only to that produced by hearing him, that one was in the presence

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of a great master. Whatever may be Tyndall's claims as an original discoverer, it is undoubtedly as an expounder of the results of original work carried on by himself or others, and as a most fascinating experimentalist, that he is best known to the present generation.

It was in 1856 that Tyndall, in conjunction with Prof. Huxley, first entered on his scientific observations on the formation and nature of the movement of glaciers. Vigorous in body no less than in mind, mountaineering possessed a special attraction for him, and he became hardly less famous as a mountaineer than as a physicist. Among his many Alpine feats he had the distinction of being the first Englishman who ascended the Weisshorn, and his Alpine excursions led to the publication of some of his most popular books—'Glaciers of the Alps,' 1860, written in conjunction with Professor Huxley, though the latter tells us his share in it was small; 'Mountaineering,' 1861; and 'A Vacation Tour,' 1862. His love of mountains was keen, and when worried and exhausted by work in town he would fly to his loved hills, and never failed to find among them renewed energy and strength.

It would ill become me to attempt to speak of the value of his scientific writings, of which "Heat considered as a Mode of Motion," "On Sound," and his researches "On Diamagnetism and Magne-crystallic Action" are perhaps the most important. One of his principal works, his "Researches on the Reactions of Simple and Compound Gases and Vapours to Radiant Heat," bears largely on climatology, and so may be said to be related to our professional work.

For many years the country had the benefit of his services as Scientific Adviser to the Board of Trade and to the Elder Brethren of the Trinity House. His independence of character and courageous outspokenness led him occasionally into differences with his official masters, and the latter post he resigned in 1883, in consequence of his dissatisfaction with the manner in which the researches into the illumination of lighthouses were carried on.

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When in 1887 he finally retired from the Directorship of the Royal Institution, which he had held since the death of Faraday in 1867, he was entertained at a farewell dinner at Willis's Rooms, which will be ever memorable from the galaxy of science which surrounded the tables.

Neither time nor space will permit me to enter on his friendship with Carlyle, whose writings had a powerful influence on him when young, and with whom he subsequently became on most intimate terms, and for whom, as Professor Huxley tells us, he had an almost filial devotion. It was of Tyndall that Carlyle is reported to have said, "What greater reward could I have than to find an ardent young soul unknown to me, and to whom I was personally unknown, thus influenced by my words?"

During his whole life Tyndall showed himself a true follower of science for science' sake, and never attempted to use his position, knowledge, and ability for gain. After his successful lecturing tour in America in 1873, he with noble generosity devoted the sum of 13,000 dollars, which remained after paying the expenses of the tour, to the foundation of a fund for aiding students engaged in scientific research at certain of the universities of that country.

In the year following his American tour Tyndall had the high honour of being President of the British Association, and it is curious after the lapse of twenty years to look back upon the uproar to which his address at Belfast gave rise in the orthodox world. Tyndall was attacked as a materialist—nay, even as an atheist by those who probably had never read his address, or other addresses which he had published in 'Fragments of Science,' which to me appear to contain very high and reverent thoughts. For instance, he says, "The phenomena of matter and force lie within our intellectual range, and as far as they reach we will at all hazards push our inquiries. But behind and above and around all, the real mystery of the universe lies unsolved, and, as far as we are concerned, is incapable of solution. Fashion this

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mystery as you will, with that I have nothing to do. But be careful that your conception of it be not an unworthy one. Invest that conception with your highest and holiest thought, but be careful of pretending to know more about it than is given to men to know."[1]

During the last few years of his life his robust health gave way, and he suffered much, among other ailments, from insomnia, to which he had always been rather subject. To the sad incidents surrounding his death, which took place at his house at Hindhead on December 4th, I need not here allude.

Dr. John Edward Tilt was born on the 30th of January, 1815. Much of his early life appears to have been spent in France, and he received his medical education at Paris, obtaining the degree of Doctor of Medicine from the Faculté de Médecine de Paris on May 15th, 1839; he does not appear to have held any English qualification until he became a Member of the Royal College of Physicians in 1859. About the years 1848—1850 he held the position of travelling physician in the family of Count Schuvaloff. I do not know when he settled in London, probably about the year 1850; after doing so he devoted himself to midwifery and the diseases of women, and became attached to the Farringdon General Dispensary and Lying-in Charity.

Dr. Tilt was a voluminous writer on subjects connected with obstetrics and gynæcology, of which the most important were "On Uterine and Ovarian Inflammation," "On the Physiology and Diseases of Menstruation," "On Change of Life in Health and Disease," and "Handbook of Uterine Therapeutics and Diseases of Women," which last was translated into German.

In his work on Uterine and Ovarian Inflammation he held the same views that his friend Dr. Henry Bennett did with regard to the great importance of inflammation of the cervix, and did not perhaps sufficiently consider diseases of the ovaries. Dr. Tilt was an original member

[1] 'Fragments of Science,' "Matter and Force," p. 93.

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of the Obstetrical Society, in which he took great interest, and successively filled the offices of Councillor, Vice-President, and President. When filling the latter honourable post in 1877, he had conferred on him the title of Cavaliere of the Crown of Italy. Dr. Tilt was also a corresponding Fellow of the Academies of Medicine of Turin, Athens, and New York.

Dr. Tilt was a very tall, handsome man, erect and spare in figure, very particular in his dress and courteous in manner; he was universally respected and liked by his professional brethren, and much esteemed by those who consulted him. In later life he suffered from some pulmonary troubles, which caused him to spend part of the year on the Riviera, and he had for some years retired from London professional life. His death occurred at Hastings from cerebral hæmorrhage on December 17th.

Arthur Thomas Myers was a member of a distinguished literary family. His father, the incumbent of St. John's Church, Keswick, was a friend of Deans Stanley and Milman, and other notable theologians, and the author of two works, 'Great Men' and 'Catholic Thoughts,' both of which were highly thought of by good judges. His brothers, Ernest and Frederick, have gained high repute in literary circles.

Arthur Myers was born at Keswick in 1851, and educated at Cheltenham and Cambridge, being a classical scholar of Trinity; he graduated in Arts in 1873, taking a first class in the classical and a second in the natural science tripos. His fondness for science led him on to the study of medicine, and he joined the Medical School of St. George's Hospital, qualifying as Licentiate of the Apothecaries' Company in 1879. At St. George's he filled the post of House Physician, and subsequently became Medical Registrar; in 1881 he took the M.D. degree at Cambridge, became a Member of the Royal College of Physicians the following year, and was elected to the Fellowship in 1893.

In boyhood and early life Myers was as distinguished

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for his proficiency in manly sports as in school and university honours. He was in his school eleven, and represented Cheltenham in the Public School racquet matches; whilst at Cambridge he was captain of the Trinity eleven, and played for his University against Oxford at tennis. Gifted thus in body and mind, an eminently successful career seemed to lie before him, but the malady which fell upon him prevented this early promise from being completely fulfilled; owing to it he was prevented seeking a post on the teaching staff of the hospital, but notwithstanding this disappointment he continued to work on quietly and unostentatiously. He became a member of the Medical, Pathological, Clinical, and Neurological Societies, and Physician to the Belgrave Hospital for Children. Dr. Myers did not contribute to our 'Transactions,' although he was the author of numerous papers of interest communicated either to other societies or appearing in periodical literature. He was the writer of several articles on hysteria, hypnotism, and allied subjects in Fowler's 'Dictionary of Practical Medicine' and Tuke's 'Dictionary of Psychological Medicine.'

Although for some months before his death his malady had increased, and compelled his retirement from the post of Physician to the Belgrave Hospital, and from professional work, his many friends were unprepared for his sudden death, which occurred on January 10th.

Dr. T. Cranstoun Charles was one of several brothers, all of whom have distinguished themselves in life. He was born at Cookstown, county Tyrone, where his father, Dr. David Charles, was in practice. His early education was gained at the Cookstown Academy, where his school career was one of distinction. Subsequently he proceeded to Belfast, and graduated at Queen's College in 1869, obtaining first-class honours and a gold medal, as well as twice carrying off the Peel Prize Essay.

After graduation Dr. Charles acted as Assistant Lecturer at Queen's College in Chemistry and Chemical

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Physics, and became Resident Physician to the Fever Hospital in Belfast. About this period Dr. Charles visited the Medical Schools of Vienna and Paris, and in 1875 joined St. Thomas's Hospital Medical School as Demonstrator of Physiology. In 1876 he served with the volunteer ambulance during the Turco-Servian war, and returning to St. Thomas's Hospital held there first the post of joint Lecturer on Anatomy and Physiology, and then that of Lecturer on Practical Physiology, which post he held at the time of his death.

Dr. Charles was a frequent contributor on scientific subjects to the daily and weekly press, and communicated numerous papers on various subjects to the scientific journals, and to Mackenzie's 'Dictionary of Chemistry.' His principal work was his 'Handbook of Physiological and Pathological Chemistry,' published in 1884, of which a second edition appeared in America three years later. I need not here allude to the circumstances under which Dr. Charles was found dead in his bed on January 24th, in the forty-fifth year of his age.

Professor Theodor Billroth was elected one of our Honorary Foreign Fellows in 1876. This illustrious surgeon was born at Bergen, in the island of Rugen, where his father was a Lutheran minister, on the 26th of April, 1829. His medical studies were carried on in the Universities of Greifswald, Göttingen, and Berlin, at which latter University he graduated as M.D. in 1852. After spending some time in visiting the Medical Schools of Vienna, Paris, and this country, he returned to Berlin, becoming Assistant in the clinic of Professor von Langenbeck, and in 1856 qualified as Privatdocent in the University of Berlin. Here his reputation rapidly increased, and in 1860 he accepted the Chair of Clinical Surgery in the University of Zurich, and Director of the Surgical Wards in the hospital attached to it. At Zurich he won the highest reputation both as an operator and lecturer, and was chosen in 1867 to fill the Chair of Surgery at Vienna. In this position Billroth's fame rapidly increased, and for many years before his death

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he may be said to have been the famous surgeon of his time. Billroth's literary activity was enormous; the number of books, papers, and monographs, all of great value and dealing with a vast variety of subjects, which he wrote is astonishing. He may be considered one of the first exponents of modern surgical pathology; his 'Lectures on Surgical Pathology and Therapeutics' have become the handbook for surgeons all over the world.

Three years after his appointment to the Vienna chair the Franco-German war broke out, and Billroth volunteered his services, and took charge of the military hospitals at Mannheim and Wissenberg. His experiences while thus occupied bore noble fruit, and have influenced in no small degree the subsequent progress of surgery.

As an operator Billroth's fame will endure, for he was the first surgeon to excise the larynx for cancer, 1873, and to resect the pylorus for cancer in 1881;[1] but great as these operative triumphs were, they do not appear to me to give him so high a title to future fame as the influence his teaching, throughout the thirty odd years that he filled the professorial chairs at Zurich and Vienna, had on surgery.

Professor Billroth was a frequent visitor to this country, where he had many friends; in the interesting introductory lecture to his Lectures on Surgical Pathology and Therapeutics Billroth does full justice to British surgery, saying, "From the time of Hunter to the present time English surgery has had something of grandeur and style about it."

In Vienna Professor Billroth occupied a foremost place both in scientific and general society; honours and distinctions were bestowed on him by all the more prominent scientific and literary societies of Europe. The Emperor in 1887 conferred on him the title of Hofrath, and made him a life member of the Austrian Upper Chamber, an honour which has seldom been conferred on a member of our profession. He was an accomplished speaker, and not

[1] The pylorus had been resected first by Pean in 1879.

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infrequently took part in the debates, always speaking in advocacy of peace and social progress.

Outside his profession Billroth's principal interest centred in music; he was an accomplished performer both on the piano and violin, an intimate friend of Strauss and Brahms, several of whose compositions were first heard at the musical gatherings which Professor Billroth delighted in holding at his house.

For two years or so before his death his health had been failing; he suffered from cardiac weakness and bronchial catarrh, to relieve the latter he had gone to spend some time at Abbazia, near Fiume, where he died suddenly at midnight of the 5th of February, in the sixty-fifth year of his age.

In conclusion I have to return my best thanks to the Honorary Secretaries for the kind assistance they have accorded to me since I was so unexpectedly called upon to perform the duties of President, and more especially have I to thank our Resident Librarian for the great help he has given me in many ways, but more particularly in preparing the obituary notices; to him also the thanks of the Society are due for the zeal with which he has discharged the duties of his office, and the able manner in which he has conducted the business relationship between the Society and its numerous tenants.

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DISEASE OF MANY JOINTS, PROBABLY OF SYPHILITIC ORIGIN.

BY

ANTHONY BOWLBY, F.R.C.S.,

ASSISTANT SURGEON TO ST. BARTHOLOMEW'S HOSPITAL.

Received April 10th—Read October 24th, 1893.

G. M—, æt. 16, first came under the care of Mr. Morrant Baker in St. Bartholomew's Hospital on November 20th, 1882, on account of ulceration of the left leg.

His history was that he had been in good health until three years before admission, and that about that time he had an attack of synovitis of the left knee for which he was under treatment in the London Hospital for two or three weeks. About a year later swellings began to form on his shin bones, and after several months one of these swellings burst, and ultimately some bone came away. The open sore thus formed had not healed.

On examining the patient there was found an irregular ulcer extending along the shaft of the tibia for a distance of about two inches. The edges were heaped up, and the

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base of the ulcer was formed by rough, bare bone. The tibia itself was thickened.

On the middle of the right tibia and a little to its inner side was a hard nodular swelling which was neither painful nor tender. There was no other evidence of congenital syphilis, the eyes and teeth being normal.

The patient was treated with iodide of potassium and mercury, and on December 16th some of the rough carious bone was chiselled off the left tibia. This operation was repeated on February 28th, and by March 30th the leg was healed. At the time of operation the bone was found to be hard and dense.

Rather more than a year later, on June 26th, 1884, the patient returned to the hospital. The scar on the left shin had broken down, and the right shin also was now ulcerated, the right tibia being enlarged, and rough carious bone being exposed in the floor of the ulcer.

The treatment adopted was the same as that which had been found efficacious formerly, but the ulcers proved most intractable, and it was only after six months' rest in bed and several gouging operations that the shins again healed.

The patient was then sent to the convalescent home, and whilst there, for the first time since he had been under notice, the joints began to show signs of inflammation, and a sharp attack of synovitis of the left knee and of both elbows necessitated his return to the hospital. The affected joints contained a good deal of fluid and were a little painful, but under treatment by Scott's dressing the fluid was quickly absorbed, and the patient was again discharged in March, 1885.

On September 1st of the same year he was again admitted. He said that he had noticed swelling on the left side of his forehead and on his right arm for about three weeks, and that his shins had broken out again soon after he was last discharged.

Examination showed a swelling attached to the right humerus about its centre, on the outer side of the arm.

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The skin over it was red and hot, and the swelling itself fluctuated. The bone around felt thickened. Over the left eyebrow was a swelling as large as a walnut, firm, and attached to the subjacent bone. In addition to the ulceration of the shins and the thickening of the tibiæ there was a good deal of effusion in each knee, and some stiffness in the movement of these joints. There was also effusion in the left elbow. The affected joints gave no pain.

On September 4th pus and caseous matter were evacuated from the swelling on the right humerus and from that over the eyebrow. In each case bare and rough bone was exposed.

The general health of the patient was very bad. He was feeble and emaciated, and had continuous slight pyrexia. The sores on the shins continued to discharge, although from time to time they healed in places.

In December the patient was troubled with cough, and effusion occurred in each pleura. The liver and spleen became much enlarged and caused a general swelling of the whole abdomen. The enlargement was evidently due to amyloid disease.

The incisions over the humerus and frontal bone did not heal, and at the end of January, 1886, a swelling appeared over the left acromion and another below the body of the lower jaw on the left side.

On February 15th albumen was found in the urine for the first time, and on February 22nd the patient died.

A post-mortem examination showed the following conditions:

Head.—A small ulcerated aperture was seen over the inner angle of the left orbit, leading to bare bone. On reflecting the scalp some cheesy, gummatous-looking material was found around the bare bone. Over the posterior part of the right parietal bone, near its inferior angle, was a soft mass of gummatous-like structure about the size of half a walnut, and in connection, below, with rough and bare bone. A similar but smaller and

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firmer mass was found over the left parietal bone: part of it was removed for microscopical examination. On removing the calvaria the membranes were found adherent opposite the largest gummatous mass, and the inner surface of the left frontal bone, beneath the ulcerated aperture already mentioned, was in a carious state over an area of about half a square inch. The outer table at the same spot was roughened, pitted, and ulcerated. The brain appeared natural.

Lower jaw.—Some caries of outer table opposite incisors, and of inner table opposite second molar.

Throat.—A considerable amount of cicatricial tissue was discovered in the posterior wall of the pharynx, close to the upper opening of the œsophagus.

Thorax.—Heart and pericardium, lungs and pleuræ quite natural.

Abdomen.—Greatly distended. The peritoneal cavity contained about four or five pints of clear ascitic fluid. The greater part of the abdomen was filled by an enormously enlarged liver and spleen.

The *liver* weighed 10 lbs. 9 oz.; its surface was scarred, especially on the left side and behind, and was puckered and contracted as though by condensation of fibrous tissue; its edges were rounded. Its section was firm and of the colour of flint, being mottled. The addition of iodine gave a marked amyloid reaction. The liver contained no gummata, nor did its section exhibit traces of healed gummata in any part. The cicatricial tissue did not penetrate deeply into the gland.

The *spleen* was enormously enlarged. Its shape was peculiar, for the notch was quite obliterated, and the whole gland was an evenly-shaped oval mass. The surface was neither puckered nor scarred. The section was firmer than natural, yet not very firm; the colour was unusually dark, and there was no indication of amyloid material to the naked eye, and no "sago-grain"-like appearance. The addition of iodine did not give any amyloid reaction.

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The *kidneys* were large and pale, but did not give the amyloid reaction to iodine.

The *intestines* were slightly amyloid in patches.

Stomach, bladder, and *pancreas* normal.

On microscopical examination much amyloid degeneration was found in the liver and spleen, and very slight amyloid change in the Malpighian capsules of the kidneys.

Right upper extremity.—On the outer side of the right arm was an irregular ulcerated aperture in the skin exposing bare and rough bone. The right humerus after removal showed the following conditions. The articular cartilage of the head was thinned in almost its whole extent, and of a bluish colour. On the posterior aspect of the head, near the anatomical neck, was a deeply-cut groove, extending for an inch or more towards the centre, after which it turned towards the great tuberosity, and ramified over the greater part of the posterior surface of the head. The posterior portion of the articular surface looked as if portions of cartilage had been gouged away, so as to leave irregular tracts with crescent-like margins. Islands of cartilage here and there remained intact. At those parts where the cartilage was most deeply destroyed the ulceration had involved the bone, and it had undergone the same gouging process as the cartilage. Crossing the bone, and closely attached to it, was a thin membranous layer. When this was peeled off, the bone was found to be rough and softer than usual. Microscopically this membrane was cellular, and was continuous with the synovial membrane, fading gradually as it approached the centre of the cartilage. In other parts the cartilage was thickened and lumpy, its thickness being at least twice as great as that of normal cartilage. The synovial membrane was more vascular, and was thicker than usual, but otherwise appeared to be normal. The shaft was thickened and misshapen by the deposit of porous new bone. The new bone was in parts tolerably firm, but in other parts was soft and crumbling. Beneath the periosteum in some parts were deposits of inspissated

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pus. The lower two-thirds of the shaft were more affected than the upper third, and at the centre of the shaft, at a place corresponding to the external ulceration, there was considerable destruction of bone with the formation of a deeply-excavated cavity.

Right elbow-joint.—There was a considerable quantity of thick synovial fluid. Part of the cartilage of the trochlear surface of the humerus had been converted into fibrous tissue, but on the anterior part of the same surface there was a nodular proliferation such as is met with in osteo-arthritis. The cartilage of the capitellum was fibrillated and pitted, but the bone was not exposed.

The posterior surface of the right olecranon fossa was rough and carious, and the periosteum was separated by broken-down inflammatory products of a caseous nature. On the articular surface of the olecranon the cartilage was in part destroyed, and the bone exposed and roughened. The articular surface of the coronoid process was in a similar condition, and between the olecranon and the coronoid processes the bone was grooved longitudinally in the manner commonly seen in osteo-arthritic joints. The shafts of the radius and ulna were healthy.

Left upper extremity.—The left shoulder-joint was next removed. It contained some inspissated synovial fluid. The cartilage of the glenoid cavity was normal, and the scapula did not seem to be diseased.

The shaft of the humerus was normal, but the head presented appearances similar to those seen in the other shoulder. An examination showed that the articular cartilage was thinned over a large extent of its surface, the thinning being less marked at the centre than towards the circumference. The thinner portions were of a bluish colour. At the lateral and posterior margins of the head, near the anatomical neck, were deeply excavated tracts. The lateral excavation commenced at the margin of the head and extended forwards towards the centre of the articular surface. The base of the excavation was covered by a fibrillated membrane, which was continuous with the

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thickened synovial membrane of the joint. A process of the synovial membrane extended forwards and accurately fitted into the excavated surface, though it was not adherent to it. The excavation on the posterior surface of the head was more irregular, and had exposed roughened bone. From the lower portion of this surface the excavation extended downwards for a short distance along the shaft into the substance of the bone itself. The synovial membrane was thickened and fringed as in cases of osteo-arthritis.

The left elbow-joint and the left radius and ulna were normal.

Lower extremities.—The shins were covered with scars which were adherent to the bone beneath. The tibiæ were thickened and covered with porous new bone. From the front of each tibia a considerable quantity of bone had been removed. The ankle-joints and the joints of the feet were normal.

The right knee-joint contained a considerable quantity of viscid fluid, and on being opened showed the following conditions. The synovial membrane was everywhere thickened and hypervascular. The patella was surrounded by a mass of fringes, some very pedunculated, others almost sessile; some large, and resembling masses of fat, others delicate and filamentous. The external condyle presented a deep groove, which runs antero-posteriorly for about an inch and a quarter. The groove extended down to and involved, but did not expose, the bone, which was covered by a membrane similar to that which lined the "ulcerated" or grooved portions in the shoulder-joint. Towards the upper and anterior part the groove became shallower, and an island of cartilage remained intact. The cartilage on the upper part of the condyle was rough and fibrillated, and greatly increased in thickness. On the upper part of the internal condyle was a large nodular outgrowth of cartilage about an inch in length. On the most convex part of the same condyle was a small mass of fibrillated cartilage. With these exceptions the rest of

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the cartilage on this condyle was normal. The cartilage of the patella was rough and fibrillated, whilst that covering the upper extremity of the tibia was slightly roughened and fibrillated on the surface. The ligaments were normal.

Left knee-joint.—The cartilage of the external condyle near to the intercondyloid notch was deeply gouged in the manner already described, as seen in the opposite knee. The destructive process had extended in a somewhat serpiginous manner, and had left islands of cartilage intact, but almost the whole of the cartilage of the anterior surface of both the external and the internal condyles had been removed, and much of the subjacent bone excavated. That part of the articular cartilage which had, during life, been in contact with the tibial tuberosities was for the most part natural, but in places was a little fibrous and thickened by the growth of small nodular masses. The bottoms of the grooves were everywhere covered by a membrane, as was the case in the opposite knee. At the edges of the condyle were nodular outgrowths of cartilage, such as are commonly seen in osteo-arthritis; none of them were of large size: the external condyle was "lipped" in a very marked manner. The cartilaginous surface of the patella was rough and fibrous, but there were no outgrowths round the edge. The cartilage of the tibia was slightly roughened, and at one spot over the internal condyle, had been replaced by a soft mass of pulpy tissue, which intervened between the free surface and the subjacent softened bone. The cartilage covering the outer articular surface of the tibia was normal except beneath the external semilunar cartilage, and here its surface was fringed and rough, and in parts slightly worn away. At its edge there was a little nodular thickening, but no definite lipping.

A microscopical examination of the affected joints showed the following conditions. A section through the articular bone and its cartilage exhibited a very extensive enlargement of the cancellous spaces, with a corresponding destruction of the bony trabeculæ. The place of the bone

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which had been destroyed was occupied in part by masses of fibrous tissue rich in cells, and in part by a more purely cellular growth. There had evidently been a rarefying osteitis with formation of fibrous tissue in the cancellous spaces.

Immediately beneath the cartilage, where the bone had been destroyed, there was an extension of the cell-growth into the hyaline matrix, with destruction of the latter to a varying extent in different parts. Here and there were seen small excavations in the cartilage, whilst in other parts the whole substance of the cartilage had been destroyed.

The place of the cartilage thus destroyed was taken by fibrous tissue, which was continuous with that found in the enlarged cancellous spaces of the bone; and where the deep pits in the cartilage extended the whole of its depth and involved the bone, the thin membrane alluded to had evidently been formed by the same inflammatory process which had been in progress in the subjacent bone.

The case here recorded is, taken as a whole, unlike other cases recorded in English literature, and I am not acquainted with a similar one published abroad. Indeed, the peculiar characters of the morbid changes, the multiplicity and extent of the joint lesions in the case under consideration, and the concurrence of extensive bone lesions seem to place it in a class by itself.

With regard to the nature of the affection, I am disposed to regard it as syphilitic, and due to an inherited disease. The points in favour of this conclusion are (1st) the appearance of the affected bones, and especially the gummatous lesions in the calvaria, and the caries and thickening of the humerus; (2nd) the occurrence of extensive scarring in the pharynx, evidently due to previous ulceration; (3rd) the scarring and thickening of the capsule of the liver. I am inclined to consider that these positive evidences outweigh the absence of some of the more common lesions of congenital syphilis, but it should be mentioned that Mr. Morrant Baker, under whose

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care the patient was during almost his whole illness, was not convinced that the conditions were due to syphilis, and was rather of the opinion that the latter was not the cause of the disease. Mr. Baker laid particular stress on the failure of the usual remedies to promote at any rate some improvement, on the marked absence of any syphilitic affections of the eyes or teeth, and on the complete want of any evidence of syphilis in the brothers and sisters of the patient or in his parents. In favour of the syphilitic origin of the trouble it may perhaps be urged that the amyloid changes in the viscera were also of syphilitic origin. I think this is very possible, but in the presence of ulceration of the shins of long standing it may well be maintained that the amyloid disease was due to chronic suppuration.

But even if the disease be considered as due to syphilis it must be confessed that the character of the joint affection is most unusual.

In vol. xxvi of the 'St. Bartholomew's Hospital Reports' I have published several cases of syphilitic disease of the joints, and these fall naturally into three classes; of (*a*) synovitis with effusion, (*b*) gummata of the synovial membrane and perisynovial gummata, (*c*) syphilitic periosteitis of the articular ends of the long bones. Such cases are well recognised by writers on syphilis, as are also the still more common cases of painless effusion in the joints of children who have other evidences of congenital disease. But although this latter class of case is not uncommon in actual practice, I am not aware of any post-mortem examinations which show the actual nature of this affection; and it is possible that in such cases lesions such as are seen in the specimens from the case under consideration may exist, although probably to a much less extent. It is, moreover, noticeable that, in the patient whose case I record, the painless nature of the lesions, the passive effusion, and the absence of any easily perceptible thickening of the synovial membrane very closely simulated

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the conditions found in this well-recognised class of chronic synovitis in children with inherited syphilis. It is also a fact that in some at least of these latter cases treatment does not produce any definite improvement; and Mr. Jonathan Hutchinson, jun., in a lecture which I shall allude to, mentions a case where the effusion lasted for many years, whilst I have recently had under my care a little girl of twelve in whom three months' treatment has caused no diminution in the size of a pair of knees greatly distended with fluid.

With regard to the joint lesions I have described, I would draw especial attention to the nature of the affection of the cartilage. It is, in my opinion, quite different from that met with in any other form of arthritis. From a microscopical point of view, the fibrillation of the matrix and the distension of the cartilage capsules would seem to indicate a relationship to the changes seen in osteo-arthritis; but the more gross lesions do not bear out the similarity. Instead of being worn down by the rubbing movements of the joints the cartilage looks as though it had been removed in segments by a gouge, and it is noticeable that this destruction does not in any way correspond to the sites of the greatest pressure or attrition. The appearance of the diseased cartilage indeed suggests a gouging, cutting, or boring process, and Mr. Baker has suggested the term "terebrating ulceration" to indicate this most striking feature of the disease.

The explanation of the destructive process in the cartilage remains for consideration, and the question at once arises whether the process originated in the cartilage itself or was secondary to disease in the synovial membrane or the bone. The answer must be determined by a consideration of the conditions presented by the synovial membrane and the bone at the site of the lesions in the cartilage, and it is at once evident that there has been no direct extension of any inflammatory process from the synovial membrane, for there are no adherent processes of the latter extending into the pits in the cartilage, such

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as are found in tubercular disease, no evidence of superficial erosion, such as may be seen in acute arthritis, and no gummatous infiltration.

On the other hand, there is good evidence that the bone at the site of the cartilage lesions is diseased, and the clinical history, as far as the lower extremities are concerned, certainly pointed to affection of the bones before the joints were involved.

It has already been mentioned in the description of the specimens that the pits in the cartilage correspond in many places with pits in the bone, and a closer examination of the latter shows that there has been a rarefying osteitis. It appears, therefore, probable that the cartilage has been involved in a process of so-called "subchondral caries," which has caused an inflammation of the cartilage spreading from below towards the surface.

Mr. Baker, however, has pointed out that there may be another explanation of the pits in the cartilage, and suggests that the pressure of the very large synovial fringes, some of which indeed appear to fit into the pits, may have caused a process of absorption, which would occur all the more readily when the subjacent bone was softened by inflammation, and when the joints were kept almost constantly in one position, as were the knees for a period of several months.

But whilst I consider that the deep erosions, pits, and grooves in the cartilage are not due to a primary chondritis, I must point out that, as I have already described, the surface of the cartilage is roughened in places where there is no deep destruction of it, and the microscope shows that this roughened cartilage is broken and fibrillated as regards its matrix, and that its cartilage cells are increased in number and distend their capsules. There is, further, a growth of nodules of cartilage forming lips around the articular margins, and these are in no way due to a subjacent bone disease. These changes in the cartilage should therefore be considered as primary, and not due to disease of the bone or synovial membrane.

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Lastly, it may be pointed out that the synovial membrane itself shows no change which can be classified as inflammatory. Its thick shaggy fringes, its smooth surface, the absence of all adhesions, and the character of its secretion make it resemble in every particular the synovial membrane from a case of osteo-arthritis. There is a complete absence of anything like either gumma or tubercle.

In conclusion I would refer very briefly to any writings that seem to have a bearing on the subject.

All recent writers on diseases of joints and on syphilis have alluded to syphilitic arthritis, and have described several varieties, and it is not necessary to say of their writings more than this, that they do not describe the changes seen in the specimens under consideration. If, therefore, these are syphilitic joints, as I consider they are, they present unusual appearances.

Virchow, however, in 1884, in the 'Berliner klinischer Wochenschrift' for August 18th, describes a form of chondritis with fibrillation of the cartilage as due to syphilis, and says that such joints may simulate those of osteo-arthritis. It is, however, difficult to be sure that the specimens he saw were identical in nature with those under consideration. In my opinion, however, the changes seen by him were in all probability of the same nature as those here described.

In vol. xl of the 'Pathological Society's Transactions,' at page 67, Mr. Jonathan Hutchinson, jun., describes a specimen of disease affecting the knee-joint taken from a man aged 23, who had stricture and ulceration of the rectum, and interstitial nephritis, but no other evidence of syphilis, and no history of it. Mr. Hutchinson considered the joint to be syphilitic, and, in alluding to it in his lectures at the Royal College of Surgeons in 1892,[1] he considers it to be an example of the chondritis described by Virchow, and of the same nature as the specimens now under consideration. I would, however, point out that

[1] 'Brit. Med. Journ.,' 1892, vol. i, p. 728.

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there is hardly even a superficial resemblance between the specimens described in this paper and the joint described by Mr. Hutchinson. It is only in the fibrillation of the cartilage that they are alike. In all other respects the specimens differ widely, for in Mr. Hutchinson's case there was during life the usual fixation and deformity commonly seen in destructive disease of the knee, the joint being flexed, rotated outwards, and displaced backwards, and all movements were greatly limited. The joint itself showed a general thickening and roughening of the synovial membrane, an absence of all pedunculated fringes, the presence of extensive adhesions, and a soft mass of gumma-like material in the substance of the synovial membrane. None of these conditions were present in the joints here described; and, on the other hand, the knee described by Mr. Hutchinson showed none of the characteristic cartilage lesions to which I have alluded.

I am, however, much indebted to Mr. Hutchinson for a reference to a paper by Gies in the 'Deutsche Zeitschrift für Chirurgie,' 1881, p. 589, in which cases of thickening of the synovial membrane and the formation of fringes, with growth of nodular ecchondroses are described in connection with syphilis, and of which good drawings are supplied. I think that some of the cases described by Gies more nearly resemble my own than others with which I am acquainted, and they, together with the paper by Virchow, support the conclusion that such lesions as I have described may be due to syphilis.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. vi, p. 6.)

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DESCRIPTION OF PLATE I.

Disease of many Joints, probably of Syphilitic Origin (ANTHONY BOWLBY).

FIG. 1.—The right humerus, showing gummatous ulceration of the shaft and destruction of part of the articular cartilage.

FIG. 2.—The right knee-joint, showing the synovial fringes around the patella and destruction of part of the articular cartilage of the femur.

FIG 3.—The lower end of the left femur, showing the deeply-gouged ulceration of the cartilage of the external condyle, and the overgrowth of cartilage on the anterior surface of the bone.

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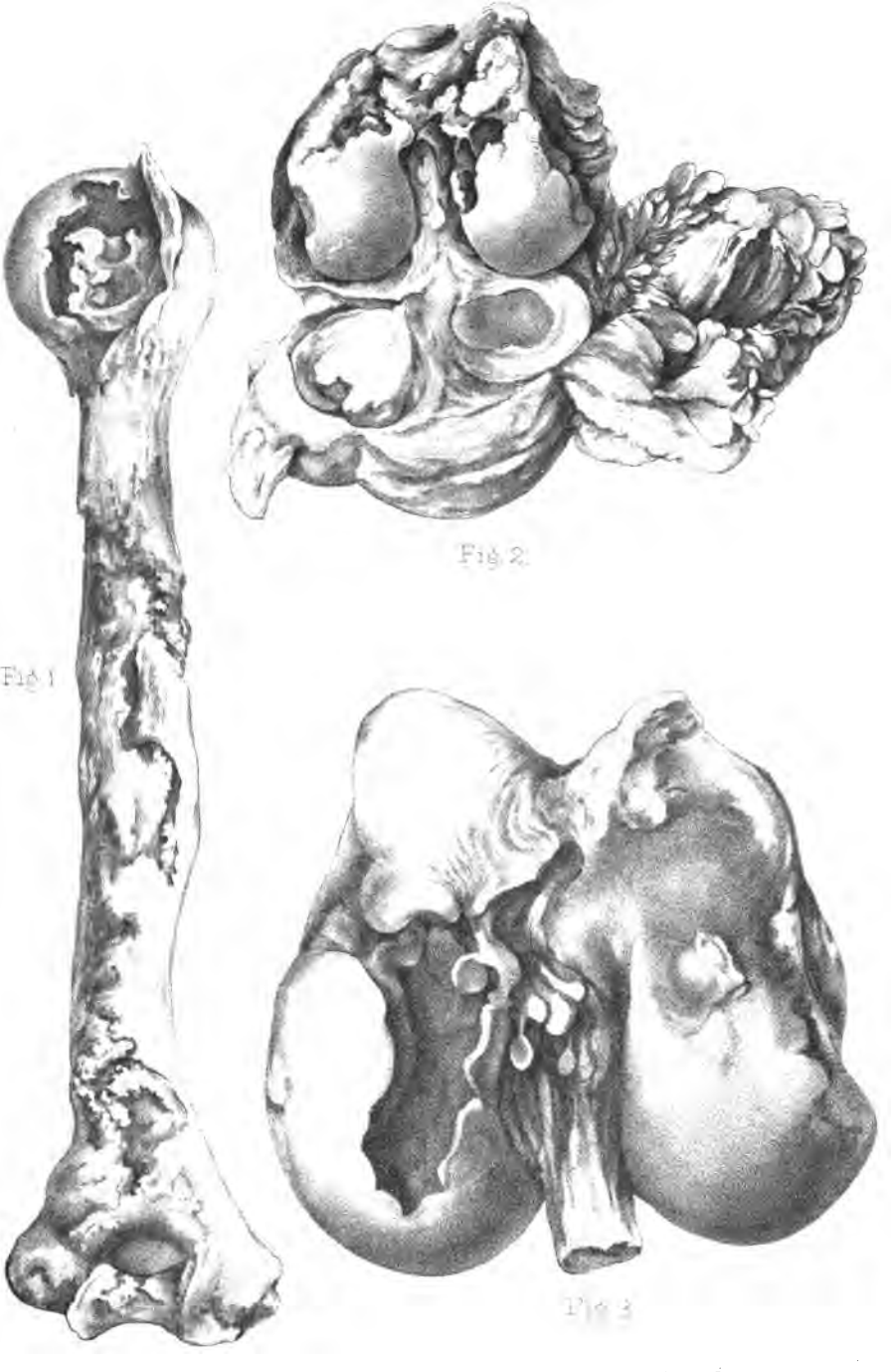

Fig 1

Fig 2

Fig 3

T. Godart del.

Danielsson & Co. lith.

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A CASE

OF

"FALSE DISSEMINATED SCLEROSIS" DUE TO MEASLES,

WITH

REMARKS ON THE OCCURRENCE OF CERTAIN WIDE-SPREAD NERVOUS DISORDERS AFTER THIS AND OTHER INFECTIOUS DISEASES.

BY

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Received April 26th—Read November 28th, 1893.

The following case, which I think must be classified as an example of so-called "false disseminated sclerosis," has been under my observation on and off, at intervals rarely exceeding two months, ever since the end of 1886, a few weeks after the attack of measles which gave rise to her nervous disorder.

Emily N—, æt. 3 years and 8 months, was first brought to the Shadwell Hospital for Children on December 29th, 1886, and was at once admitted under the care of Dr. Eustace Smith, to whom I am indebted for the use of the notes of the case while an in-patient. The mother considered herself to be a healthy woman, but she has since

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been frequently treated for migraine, which she has in a severe form. The father was described as healthy, and two other children were and have remained healthy, with the exception that all three had chicken-pox in 1889.

The patient's younger brother was taken ill with measles on November 21st, 1886, and the patient herself showed the eruption of the same disease on November 30th. Dr. H. C. Wilson, who attended her, has been kind enough to inform me that she appeared to be suffering from an ordinary attack of measles, the rash being well out, when suddenly, on December 3rd, she was seized with convulsion. She was unconscious for ten days. After coming to her senses she could not speak, and had lost all use of her limbs.

When admitted on December 29th she had a very vacant, stupid expression, and was extremely apathetic. She appeared to recognise people about her—her mother, for instance, but cared nothing for their coming or going. There was some difficulty in swallowing. The food ran out of her mouth unless it was pushed well to the back of the tongue. She was very costive. She could not sit up, though she could move her limbs feebly; there was no obvious muscular atrophy. Two days later she was reported to have taken food well, and to be free from fever. She did not speak, and the mother stated that she had last spoken on December 3rd, the day the fit came on. She could put out her hands, the left better than the right. Motions and urine were passed under her. There was no cyanosis. The skin was of a good colour. She looked well, but kept the same vacant expression. She lay in bed with the lower limbs flexed, but when they were passively extended she seemed to have very little power of flexion; she could move the toes. There was, apparently, anæsthesia of the legs, neither an ordinary touch nor a prick being followed by any response. When the abdomen was pricked she frowned slightly. She could squeeze, but not strongly, with both hands. There was general and extreme paresis of both upper limbs, and

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it was noted that "full control over the movements was not present." When raised into a sitting posture she could maintain herself in that attitude by holding on to the sides of the bed. She had to be fed by the nurse, and there was apparently some difficulty in closing the lips.

On January 4th, 1887, she was brighter, took hold of a hand held out to her, and was said to have spoken. She sat up, but could not stand. She did not appear to feel the prick of a pin anywhere in the lower limbs except in the foot; when this was pricked she "made a face" and the foot was drawn up, *i. e.* the toes and knees were flexed. The next note was made on January 13th. She could feed herself, moving her lips, masticating and swallowing slowly. She could not stand. She could feel the prick of a pin anywhere, but sensation appeared to be everywhere dulled, though there was no distinct area of anæsthesia. The knee-jerks were "easily got." There was no ankle-clonus. Though she could not stand, she could move her legs and toes slightly when seated on a chair.

On January 18th it was noted that the difficulty she found in feeding herself appeared to be due not so much to want of power as to inco-ordination. She grasped a piece of bread pretty firmly, but in carrying it to the mouth the movements were uncertain. When held in the upright attitude she made forward movements with both legs, but with the right better than with the left; on this day she tried to get on her knees. Sensation seemed to be returning in the limbs.

By February 15th there had been considerable improvement; she was able to walk, and talked a little, using single words. Thereafter there was little or no improvement while she remained in hospital, which she did until April 3rd. She would stand wherever she was put, leaning against any object, such as a chair, or the high fireguard, holding on with her hands and putting very little weight on the legs.

After her discharge from hospital she continued to improve, but very slowly. At the end of two years she

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was able to walk, but was easily fatigued. There was a fine tremor of the hands and upper limbs, aggravated when she was told to perform some definite act, as picking up a pin, or lifting a glass to her lips (with both hands). She was very much troubled at a later date by chronic diarrhœa and prolapsus ani, but with that exception her general health has been, on the whole, remarkably good, and her growth does not seem to have been retarded. Her mother thought that she was quite as intelligent as other children, but she appeared to be dull, and when an in-patient in 1891, on account of the prolapsus ani, she was considered by the nurses to be half-witted. She goes to school, but is still in the lowest standard. On the whole I think that there is certainly some mental deficiency, though this is not so great as strangers, judging from her very vacant expression, commonly suppose. She has undoubtedly gained considerably in the power of speech. Until she was six years old she spoke very slowly, and appeared to have difficulty in finding words to express her meaning. Speech was syllabic: she would tell me that she was "bet—ter." In this respect there has been some improvement; her utterance in November, 1891, was very deliberate, but hardly to be called scanning, and she did not seem to hesitate for a word. She could walk fairly rapidly, and as far perhaps as other children of her age, but her gait was of the spastic character, the legs were adducted, and she took very short steps. Owing to imperfect flexion of the knees the toes were very liable to catch as the foot was brought forward, whence many falls. There was no inco-ordination, but the movements of the lower limbs, when she was lying or sitting down, showed a small amount of irregular coarse tremor, and the head was unsteady. There was no paralysis of the arms, but the finer movements were not well performed, owing to a slight tremulous uncertainty. She could drink without difficulty, though the act was slowly performed, and swallowing was noisy; the cup was not rattled against the teeth, but there was some slow tremor of small excursion.

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There was decided rigidity of the lower limbs, but the deep reflexes were only slightly exaggerated; the knee-jerks were very easily obtainable, and very free—in fact, exaggerated; there was no ankle-clonus, but front-tap contraction was present. Tactile sensibility was unaffected. There was no rigidity of the arms. Micturition was normal; and the prolapsus, to which reference has been made, appeared to be of the ordinary type—the bowel came down when she had a motion, and the stools were slimy. The condition was recovered from, under the use of a castor-oil mixture.

A note of her condition was taken about six years after the attack of measles. The deep reflexes were distinctly increased in the lower limbs. Knee-jerk was exaggerated, and on flexing the foot on the leg three or four clonic contractions occurred; front-tap contraction was elicited very easily, and one percussion was followed, as a rule, by three or four contractions. An effort to perform any movement revealed a little coarse tremor of the hands.

On April 12th, 1893, when she was ten years old, her mother stated that she was still very backward at school, could only read little words, and could write "hardly at all." Owing to the unsteadiness of the hands she cannot be trusted to help her mother, and the attempt to, for instance, wash a cup, leads to much shaking, not only of the hands, but also of the head, and is liable to end in the destruction of the cup. There is slight tremor on attempting to pick a pin off the table, and it is aggravated when she attempts to lift a tumbler full of water. She walks rapidly and steadily, but with short steps, and the mother states that she has walked as far as six miles recently. She is very easily upset, and has had so many falls that she is no longer allowed to go into the school playground.

The symptoms observed in this case present a general resemblance to those of disseminated sclerosis. The presence of tremor, aggravated by attention, the scanning

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speech, the dull, heavy expression of the face, with drooping lip, half-open mouth, and general air of stupidity, the spastic gait with exaggerated deep reflexes, are points of agreement; on the other hand, no nystagmus has been observed at any time in the course of the case, and the disease has not been progressive, but has shown a steady tendency to improve.

Although cases of a similar character have been recorded as occurring after other acute infectious diseases, I have not been able to find a parallel case after measles. There are, however, a considerable number of anomalous widespread nervous affections occurring as complications or sequelæ of measles on record. Some of these present points of resemblance to the case of E. N—. Dr. Colcott Fox has recorded a case in which a girl aged four and a half, when beginning to recover from an eruptive illness which the mother stated to be measles, became vacant and unable to sit up. Two months later there was marked inco-ordination of the head and limbs, nystagmus, slow laboured speech, and vacant expression. The reflexes were not exaggerated. The limbs were weak, and there is no mention of rigidity. The child improved slowly, and when discharged eight months after the onset of the illness she was almost well. In a case observed by Schepers the patient, a girl aged eight, suddenly became comatose on the fourth day of a mild attack of measles. When she recovered from the coma, three days later, she was unable to speak. The movements of the upper limbs were "choreiform;" she could not stand owing to ataxia, and there was great impairment of memory. A fortnight later speech was slow, heavy, nasal, and brief. She could stand resting against a table. Later she began to walk, at first with the aid of sticks, but her gait was uncertain, and resembled that of a person with locomotor ataxy. The Pendlebury *Reports* (1883) contain a brief note of the case of a girl aged four, who when taken out of bed after an attack of measles in August, 1882, was found to be unable to walk. When

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admitted on December 4th she was unable to bear the whole of the weight of her body. The knee-jerk was increased, and front-tap contraction was present, but not ankle-clonus. She improved under treatment by faradisation, but when the last note was made, about six months after the attack of measles, though she could stand alone for a short time, she swayed from side to side; she could walk with help, throwing the legs forward and bringing them down on the heels; the knee-jerks were much exaggerated, but there was no ankle-clonus.

A case which has an important bearing on the subject is that recorded by Dr. Thomas Barlow in the 'Medico-Chirurgical Transactions' (lxx, p. 77). In that case the patient, an adult male, first presented distinct nervous symptoms on the fourth day of the exanthem, and died about thirty-six hours later, apparently from asphyxia. Microscopical examination made by Dr. Penrose showed wide-spread acute myelitis of the disseminated type, the maximum affection being in the upper dorsal region, where the cord was diffluent. In other regions of the cord, however, the parts most affected were the anterior cornua. The lesions were all vascular—hæmorrhages and intense distension—and no changes were found in the nerve-cells, fibres, or supporting tissues of the cord. Similar changes were found in the medulla oblongata.

The nervous affections which have been observed as the more or less enduring consequences of measles fall into three classes:

(I) Hemiplegia.

(II) Cases of limited muscular atrophy, of the type of infantile paralysis. (To these conditions I do not propose to refer more particularly here.)

(III) Cases of widely diffused nervous affection, lasting for varying periods:

(*a*) Acute disseminated myelitis.

(*b*) Cases presenting at a later date symptoms resembling disseminated sclerosis (false disseminated sclerosis).

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(*c*) Cases in which, with some symptoms similar to those of the preceding group, the most prominent symptom is inco-ordination.

(*d*) Cases of "extensive, ascending, diffuse, or disseminated" paralysis, resembling diphtherial paralysis.

The points to which, in commenting on the case here recorded, I would especially draw attention are two:

In the *first* place, that, as is well known, a similar complex of symptoms is occasionally observed after many other acute infectious diseases; and

In the *second* place, that after measles and other acute infectious diseases, a condition to which the term pseudo-ataxy has been applied is sometimes observed, and that this condition, from which recovery occurs, is probably related to "false disseminated sclerosis" in the sense that the one is a minor form of a similar order of lesion.

With regard to the first point—the occurrence of cases of false disseminated sclerosis after various acute infectious diseases—it would be tedious to cite individual cases recorded in literature. It will suffice to say that the symptom-complex has been observed after scarlet fever, variola, typhoid fever, whooping-cough, influenza, and diphtheria.[1] An examination of the cases under each of these diseases to which references are given, and of the collections made by Whipham and Myers, and by Marie, is sufficient to prove this point.

The clinical histories of the cases in which this complication arises present a considerable degree of similarity. Symptoms pointing to a grave involvement of the nervous system are, as a rule, first noticed after defervescence; but in not a few cases the patient has suffered from convulsions during the acute stage, or a peculiar condition of stupor or somnolence has been noticed at that period. The clinical history of "pseudo-ataxy" is not very dissimilar, the ataxic paralysis, which affects the lower limbs

[1] To this list ought, perhaps, to be added erysipelas, pneumonia, and ague.

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in most cases more distinctly than the upper, being observed when the patient first attempts to get out of bed.

It appears to me to be quite impossible to draw any distinction between the symptoms presented by these cases of "pseudo-ataxy" and those not infrequently observed in the course of diphtherial palsy, indeed the best examples of "pseudo-ataxy" are to be seen after diphtheria.

Further, I would submit that the difference between these cases and those of well-marked "false disseminated sclerosis" is one of degree rather than of kind. Improvement in "pseudo-ataxy," though it may, after it has once begun, progress for a time with great rapidity, in some cases becomes greatly retarded at a later date, so that such patients cease to seek medical treatment while there still remains some defect in speech, or some uncertainty or weakness in the movements of the lower limbs.

It is common to assume that diphtherial paralysis, if it do not cause death at an early stage, always ends in recovery, that is to say that the parts paralysed recover their function. This, however, is not always the case. Some permanent affection of the muscles of deglutition or phonation remains in certain patients. In others there remains some disorder of the motor functions of the lower limbs, spoken of commonly as paraplegia or paraparesis, but in which some degree of ataxia exists. Further, it is important in this connection to note that cases of the type of "false disseminated sclerosis" have been observed after diphtheria (Stadhagen, Schoenfeldt).

Finally we have observations of acute disseminated myelitis terminating rapidly in death, by Westphal in smallpox, and by Barlow in measles. Westphal has suggested, as Barlow pointed out, that a slighter degree of mischief than that found in acute disseminated myelitis might give rise to changes which would result in a clinical condition resembling disseminated sclerosis.

In conclusion, I would lay stress on the following points:

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The occasional occurrence after all, or nearly all, the acute infectious diseases observed in this country of cases presenting symptoms indicating a wide-spread affection of the nervous system.

The fact that the grouping of the symptoms renders the cases, whatever may have been the infectious disease to which they were secondary, strikingly similar to each other and to diphtherial paralysis.

The fact that occasionally in the pyrexial stage of acute infectious diseases, or during convalescence, there is developed, usually with rapidity, a profound affection of the nervous system, characterised usually by loss of consciousness; the patient, if he survive, being found on recovery of consciousness to be bereft of speech and to be suffering from extensive paralysis, paresis, or ataxia. From this condition he may recover completely, or he may develop symptoms resembling those of disseminated sclerosis, but with this difference that the disease is not progressive but rather regressive. In the cases fatal at an early stage, the changes in the central nervous system appear to have been mainly or primarily vascular; and the most reasonable hypothesis to account for those cases in which complete recovery takes place at an early date is that they also are due to vascular derangement. Analogy would suggest that this vascular derangement is produced by the circulation of soluble poisons.

That cases of "false disseminated sclerosis" may be observed as a sequel to diphtherial paralysis.

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(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. vi, p. 21.)

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ON A

DISSEMINATED FIBROSIS OF THE KIDNEY

FOUND IN

CASES OF INFECTIVE ENDOCARDITIS, AORTITIS, AND ALLIED DISEASES,

ITS PATHOLOGICAL SIGNIFICANCE, AND CHIEF CLINICAL FEATURES.

BY

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THE following kidney disorders, excluding those obviously due to embolism, I have found associated with infective endocarditis:

1. The tough congested kidney, wherein a general venous engorgement of the whole organ is the chief if not the only alteration visible.

2. The large mottled kidney. In this case the organ is tough and misshapen, and its congested surface is relieved by well-defined elevated patches of pale granular tissue. On section, the mottling is found to extend throughout the

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thickness of the organ; portions of the pale material protrude into the pelvis, and consist mostly of tubules blocked with swollen and degenerated epithelium. Such kidneys occasionally secrete an inordinate quantity of highly albuminous urine. One of my cases passed upwards of 100 oz. daily for several successive days; and on one occasion the water contained 1·55 per cent. of albumen.[1]

3. The small mottled kidney. Here also the organ is misshapen, and usually mottled, pink and white. Extreme toughness, small size, and puckered surface are its chief peculiarities. This type of kidney secretes little water. The urine is of a low specific gravity, and slightly albuminous.

My contention is that these varieties of kidney degeneration, at all events the two last types, are different phases of the same progressive disease.

The distinguishing feature of this disease is the presence of scattered groups of nuclear-like bodies in the loose interstitial tissues of the affected kidney, especially around the smaller arterioles and the Malpighian tufts. These bodies are deeply stained by many aniline and other dyes; and to their presence in the renal tissues the subsequent degeneration of the organ can with some probability, as I shall subsequently show, be traced. Associated with these kidney changes, we frequently find more or less extensive disease of the aorta and of the cardiac valves. We shall also find in these cases, if we search for them aright, groups of similar nuclear-like bodies between the inner and middle coats of that artery or beneath the endocardium. I have also found them, deeply stained as usual, in the adventitia and around the *vasa vasorum* of the aorta.

On the other hand, acute endocardial mischief is sometimes absent in cases of disseminated renal fibrosis, although evidence of old-standing cardiac trouble in the thickened and opaque cusps is mostly present. Aortitis,

[1] Dr. Adolphus Richardson (adopting Brandberg's method) kindly made this estimate for me.

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as noticeable to the naked eye in discoloured patches of atheroma, is constantly found, and as often overlooked. In connection with this subject I may remind my audience that the great pathologist whom we have recently seen amongst us, Rudolf Virchow of Berlin, drew attention nearly five and thirty years ago to the relationship which exists between the processes leading to arterial atheroma on the one hand, and to endocardial degeneration on the other ('Cellular Pathology,' Eng. edit., p. 362).

The nuclear bodies, their size and shape; other characters.—The nuclear bodies are small oval or roundish bodies, variable in size, but for the most part less in diameter than is a blood-corpuscle. In the densest groups the smallest sized bodies are seen, usually consorting in twos or threes within apparently one cell, while the larger ones are generally solitary and frequently without any obvious cell membranes around. Under a high power (Zeiss F, oc. 4, Abbé's condenser) they are found to consist, after staining, of two portions, namely of numerous deeply-stained granules embedded in a pale hyaline plasma. Although these bodies are found for the most part in the loose connective tissue surrounding the vessels of the regions they frequent, and although I believe that by their presence in this tissue they are directly responsible for the kidney degeneration to be more fully described elsewhere, yet they are rarely seen in the thickened fibrous tissue of Bowman's capsules or in the hypertrophied walls of the arterioles. They appear to require elbow-room to grow and thrive in, as do their unfortunate hosts.

The kidney degeneration.—The morbid changes observable in the renal structures as the outcome of this disease may be conveniently placed under two headings, according as they are apparently the result of mechanical or of purely pathological processes. In considering the mechanism of urinary secretion, prominence must necessarily be given to the renal arteries and their branches for the part they take in regulating the supply of blood by means of their muscular coats to the varying require-

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ments of the organ. But the alternate dilatation and contraction of a tubular vessel necessitate a more or less rapid change of its calibre. The renal arterioles in order to permit this variation in their sectional area are embedded in a delicate elastic tissue which readily yields to any change in size or shape. When, however, the resiliency of the perivascular tissue is impaired at various points by the introduction of rapidly proliferating corpuscles amongst its meshes, an early result must be the development of corresponding points of muscular weariness in the embedded vessel, followed by a more or less continuous diastole. As a consequence of the collapse in the system which regulates the local blood-supply, we find areas of vascular engorgement developed frequently throughout this disease. After a time in many cases the affected vessels undoubtedly recover their regulating powers at the expense of their muscular coats. Meantime, however, other groups of nuclear bodies are heaped around the necks of the Malpighian corpuscles, or are scattered over the membrane covering a tuft, or, again, pervade a Bowman's capsule. This sudden invasion of the kidney substance—for, I take it, the evidence, both clinical and pathological, points to a succession of explosive outbursts in this disease followed by periods of comparative calm—this sudden invasion of the kidney substance, I repeat, by these pathological entities must, and speedily does, entail secondary changes, both organic and functional. And so we find districts of fibrous hyperplasia developed, containing thickened capsules and shrunken tufts, and adjoining others wherein the glomerules are apparently healthy. Hypertrophy of the muscular walls of the interlobular arteries and their branches follows, partly in consequence possibly of these vascular changes, partly from the increasing thickness of the adventitia. These abnormal circulatory conditions are subversive of the orderly adjustment of the blood-supply of the kidney to the demands made upon it, and as a result we meet with evidences of over-stimulation of the epithelium, clinically

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in an excessive secretion of albuminous urine, pathologically in considerable masses of pale parenchymatous tissue, consisting mostly of swollen and dilated tubes choked with epithelial *débris*. As the disease advances, or whenever the nuclear bodies become generally dispersed throughout the interstitial tissues, a condition arises similar to that which leads to the contracted granular kidney of Bright. I need not here recapitulate the minute changes of the organ under such circumstances, as they have been repeatedly discussed at the Society's meetings by many illustrious Fellows, notably by Sir George Johnson. I may, however, be permitted to mention that the twenty-eighth volume of the 'Transactions' contains a paper by Sir James Paget on the coincidence of granular degeneration of the kidneys with old clots in the pulmonary arteries.

The disease of the endocardium and aorta which so often accompanies disseminated fibrosis of the kidney consists essentially of nests of nuclear bodies similar in size, in shape, and in physical properties, as far as I can ascertain, to those located in the kidneys. They are found beneath the inner coats of the heart and artery respectively, and they undoubtedly give rise to the structural degenerations so fully described by Virchow (loc. cit.). I have, as before mentioned, found the nuclear bodies in the adventitia of the aorta.

The question how these morbid units are conveyed, say from the aortic intima to the renal tissues, if we admit these pathological processes to be dependent the one upon the other, is a difficult one to answer. I imagine that they are conveyed directly by the blood either within a leucocyte or separately, and that they subsequently select a favorable site to penetrate the walls of the renal vessel. I feel assured that the nuclear body is quite capable of passing through the comparatively thin-walled arteriole, as I have found them occasionally between the layers of the tough elastic coat of the aorta. Are the nuclear bodies when present in kidney disorders the *fons et origo*

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malorum? If we are to believe the essence of this disease to be particulate, I think that they are so. But whether the virus lodges within the nuclear bodies themselves, or only in the highly-stained granules, I shall not venture to decide. I have been unable to find any bacilli, either stained or unstained, among the affected tissues, although fibrillar irregularities in a certain light can produce rod-like appearances which might readily be mistaken for the organisms mentioned.

Finally, as regards the chief symptoms observable in the disease. One of the most important is hæmorrhage from a mucous surface: bleeding from the nose or mouth, hæmoptysis, hæmaturia, and rectal hæmorrhage are commonly met with. The pulse tension is usually *plus*, and the heart is hypertrophied. Valvular incompetence can possibly modify this condition. Anæmia is generally severe. One of my eleven cases had only 24 per cent. of the normal number of blood-corpuscles in his vessels. Repeated short attacks of unconsciousness, with and without rigidity of the muscles, occurred in three cases. Clots in the veins occurred in two, in one of which the right subclavian was blocked. Temperature was variable.

Appendix.

In the following notes the cases have been arranged, as near as may be, with regard to the kidney-changes seen in the organs after death.

Case 1. *Congestive mottling of kidneys; early stage.*—Arthur H—, æt. 11, a schoolboy residing at Brighton, was admitted to the Sussex County Hospital on July 14th, 1893, with symptoms of recurrent appendicitis. Present attack began a fortnight previously with pain in the abdomen. Had a similar attack at the beginning of the year, necessitating his absence from school for several days. On admission patient was collapsed; face pinched; abdomen distended; tongue dry and furred; bowels not

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relieved for four days; constantly vomiting fæcal matter. In the afternoon Dr. Furner opened the abdomen and discovered adhesions in the neighbourhood of the appendix with strangulation of a loop of intestine. This was relieved, and the bowels were with difficulty replaced. The patient died about fourteen hours afterwards. At the autopsy the appendix was found to be ulcerated through, about half an inch from the cæcum. The free end was the seat of an abscess, behind the cæcum and shut off from the peritoneal cavity by adhesions. The aorta was striated just beyond the valves with patches of atheroma. The kidneys were somewhat tougher in texture than natural. Capsules separated readily. There was much congestive mottling of the surfaces of the organs.

I have added the above case to those already collected before writing this paper as the microscopic sections of the aorta and kidney show the nuclear bodies very distinctly, and what I take to be the earliest changes in the renal structures due to their presence.

Case 2. (This case has also been added since the paper was written.)—H. R—, a barber, æt. 21, residing at Brighton, was admitted on June 6th last, also suffering from abdominal symptoms. Five days ago, shortly after eating some whelks, he was seized with pain in the right iliac fossa, which soon spread over the whole abdomen. Tenderness was well marked. For two days before admission he had frequent vomiting. Constipation had persisted during the attack, notwithstanding doses of jalap and senna. He had a similar attack last January, which lasted five weeks. Family history was good. On admission the temperature was normal. Tongue dry and furred. Abdomen distended and tympanitic, with well-marked tenderness generally. Respirations thoracic in character. Heart's apex-beat one inch outside nipple-line in the fourth space; some roughness with the second sound. Basal aortic sounds feeble. Urine sp. gr. 1035, acid, no albumen.

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During the next two days the youth remained mostly in the same condition. The abdominal tenderness, however, became localised over the right iliac fossa, and the temperature was usually a degree or two above normal. The bowels were not moved, although he passed flatus *per rectum*. On the evening of the 9th he was suddenly seized with severe pain about the navel, with vomiting. The bowels were opened twice, motions formed and dark coloured, no blood. The pain gradually became more severe; the next morning collapse set in. The pulse rose to 152, thready in character. Temperature 103·2°. The patient's intellect continued clear throughout; he did not consent to an exploratory operation recommended by the staff. He died that afternoon. At the autopsy pus was found in the abdominal cavity, and there were numerous adhesions between omentum and the coils of intestines. The extremity of the appendix had sloughed away, and an opening was found passing into the cæcum, and partly blocked by an intestinal concretion. The left ventricle of the heart was hypertrophied. No valve disease noted. There was a considerable patch of atheroma in the aorta. The capsules of the kidneys were somewhat adherent, and their substance congested and tough.

Nuclear bodies could be seen between the inner layers of the elastic coat of the aorta forcing it inwards.

Case 3. *Tough congested kidneys.*—I. W. B—, æt. 67, a retired soldier, had been ailing for many months and on two previous occasions an inmate of the Sussex County Hospital with similar symptoms. Admitted February 1st, 1893. He had been almost continuously in bed for the last five months with shortness of breath, cough, and swollen legs. No history of syphilis or of acute rheumatism. On admission the patient's temperature was subnormal. Face and extremities cyanosed. Urine acid, no albumen. Heart's apex in fifth space, half an inch outside the nipple line. Cardiac dulness absent. No bruits audible. Lungs emphysematous; rhonchi general

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over the back; both bases fairly clear. Some œdema of the dorsal integument. Abdomen tense and resonant. No visceral enlargement detected. Legs hard, red, and œdematous. Penis much distended with œdema. Passed urine with difficulty, and frequently. During his stay in hospital the lividity of his face increased, the œdema became more marked, and the temperature remained subnormal. The heart's sounds meanwhile were very feeble and indistinct, and the dyspnœa was distressing. The patient died somewhat suddenly twelve days after admission.

At the autopsy the lungs were found to be emphysematous, and overlapped the heart. The latter organ was both dilated and hypertrophied. The valves of the right cavities were thickened. On the left side the mitral was greatly thickened, and the aortic cusp calcified at its attachment. The aortic valves were converted into a calcareous ring with a slit in the centre, through which water could pass in either direction with the same facility. On the endocardium below, and on the aorta above, were atheromatous patches covered with vegetations. The kidney cortex was diminished in amount, and the capsules stripped fairly well. On section the whole organ was seen to be intensely congested, and it was tougher than natural.

Numerous groups of nuclear bodies were visible in the section of the aorta.

Case 4. *Large mottled kidneys.*—Fred K—, an errand boy, æt. 16, was admitted to the Sussex County Hospital on November 8th, 1892. Two years ago had a small impacted stone removed from the urethra; subsequently had rheumatic fever with pericarditis (?). Had been short of breath since that time. On admission he was found to be suffering from pains in the joints without perceptible swelling, and from much sweating. Heart's apex felt half an inch outside nipple line in fifth space. A soft systolic murmur at apex, heard towards axilla; and a to-and-fro

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sound at base over aortic cusps. During his stay in hospital he had several attacks of epistaxis. The temperature was mostly 100° and upwards. The urine was very albuminous, sp. gr. about 1020; he passed from 60 to 80 oz. daily for some weeks. He had general œdema of the integuments, and was tapped twice for ascites. He died on February 15th, 1893.

At the subsequent examination the following conditions were noted: The body was fairly developed. There was general anasarca. Pleuræ contained two or three pints of clear serum, and the lungs were much collapsed. Heart weighed 17 oz. Pericardium universally adherent; cardiac cavities both hypertrophied and dilated. On one of the tricuspid flaps was a small white nodule with roughened surface, and the adjacent ventricular surface was coated with a small rough fibro-cartilaginous patch. On the left side both sets of valves were roughened and ulcerated along their free borders; there were also patches of roughened endocardium at the position of the foramen ovale and along the ventricular walls. The kidneys together weighed 7 oz. They were much mottled. The left was very small.[1] Cortex narrowed and remarkably white in parts. Capsule stripped readily. The right was considerably enlarged. Capsule peeled readily. The section presented several white patches of tissue extending from the cortex to the pelvis, into which some of them projected, seemingly distinct from the rest of the substance. These patches consisted mainly of dilated tubules filled with swollen epithelium and other detritus.

Case 5.—Wm. S. T—, æt. 37, was admitted into the hospital on November 23rd, 1892, in a semi-conscious state. He had suffered from pain in the head and back, with hæmaturia and vomiting, for five weeks. Present illness came on after exposure. Previous health good. On admission the temperature was subnormal. Pulse 72, cord-like. Respirations 16, long and deep. He answered a question

[1] Probably a congenital condition.

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when roused. Colour of skin good, no cyanosis. Pupils equal, somewhat dilated. He could move his limbs. No muscular twitching. Heart's apex half an inch outside nipple line in fifth space. Prolonged systolic murmur at apex. Reduplicated second sound at base. Lungs clear throughout. Urine sp. gr. 1012, alkaline, very albuminous; on one occasion it contained 1·55 per cent. albumen. An excessive quantity was passed; once it reached 116 oz. in the twenty-four hours. The patient complained of severe occipital headache with increasing dimness of sight. There were recurrent attacks of vomiting, and of rectal hæmorrhage. During his stay the head symptoms became more prominent. There was loss of sight in left eye with atrophy of disc, and paresis of rectus externus. The blood contained at this time only 24 per cent. of red corpuscles. Lung symptoms were subsequently developed; a patch of consolidation appeared at the upper part of left, and another at lower part of right lung. No tubercle bacilli were met with in sputa. The legs became œdematous through blocking of the saphenous veins a few days before death, which occurred on March 6th following. At the autopsy discrete glistening white bodies about the size of pins' heads were scattered over the pia mater of hemispheres and choroid plexuses. The left upper lobe was sharply marked off from the rest of the lung; it was of a light grey colour, consolidated and airless; bands of fibrous tissue could be traced from the surface spreading through the lobe. The substance was, notwithstanding, somewhat soft and friable. In the right lower lobe were two or three cavities. Heart weighed 22 oz.; cavities both hypertrophied and dilated. The tricuspid orifice admitted five fingers. Valves healthy. The mitral curtains contained patches of thickening, and their edges were also thickened. Aortic valves were healthy. There were many patches of atheroma along the commencement of aorta. The kidneys weighed together about 12 oz. Their contour was misshapen. Their surfaces were mottled with pale and congested

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patches. The capsules stripped easily. The pale blotches were raised above the surrounding surface, and they extended into the substance of the organs. The bases of pyramids were frayed out. The kidneys were very tough and fibrous. The omentum and the capsules of the liver and spleen were adherent to the abdominal walls. Groups of nuclear bodies are disposed around the thickened renal vessels. The uriniferous tubules were in various stages of degeneration.

Case 6. *Large pale kidneys.*—James B—, æt. 62, a labourer, residing at Brighton, was admitted September 2nd, 1892. He was then suffering from diarrhœa, vomiting, and swollen legs. The illness began two months ago. Had recently passed a large quantity of water daily. Eyesight good. Both his previous and his family histories were good. On admission very chloræmic; hæmoglobin 20 per cent. Some dyspnœa; legs œdematous. No hæmorrhages. Heart's apex in normal position. Systolic murmur heard at apex, conducted towards axilla; also a systolic murmur heard over the aortic orifice and upwards. Breathing heard faintly at lung bases. Bowels open four or five times daily. Urine sp. gr. 1012, no albumen. Passed rather more than usual quantity; on one occasion 43 oz. in the twenty-four hours. Temperature about 99·0°. He was discharged on September 26th, 1892, improved in health. On October 24th last he was readmitted to the hospital. For three months his breath had become shorter, and the legs and arms had swollen more than usual. Sight was failing. No history of rheumatism; he stated that he had had two or three attacks of jaundice (?). On admission, very sallow and chloræmic; no jaundice. Temperature normal. Pulse regular, of high tension. Tongue pale and flabby. Heart's apex in fifth space in nipple line. Cardiac dulness absent. Prolonged apical systolic murmur, conducted outwards. Rough systolic murmur over pulmonary cusps. Bases of lungs clear; some emphysema and rhonchi.

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No enlargement of liver or spleen. Legs slightly œdematous. Urine contained no albumen. He died two days subsequently. At the post-mortem examination on October 28th a considerable thickness of subcutaneous fat was found. There was some fluid in either pleura; and two or three roundish bodies, very hard and calcareous, and about the size of small peas, were attached to the membrane. The central tendon of diaphragm contained a plate of bone about three inches in diameter. The cartilage rings of the lowest fourth of trachea and at the commencement of large bronchi were calcified. Heart weighed 20 oz. It was much dilated, especially the right ventricle. The left was also much hypertrophied. Muscular tissue pale and friable. Tricuspid, mitral, and aortic valves were all thickened. The chordæ tendineæ of left ventricle were also thick and short. There were many patches of atheroma on the endocardium and along the aorta, mostly of the circinate variety. At the upper part of the aortic sinus was a concavity about half an inch deep, with atheromatous walls. Liver small; capsule thickened. Kidneys together weighed 1 lb. Pale and very tough. Capsules thick and adherent, left the surface granular; many cysts in cortex. Papillæ shortened. A large amount of fat surrounding pelves. Nuclear bodies were to be seen in the aortic intima, and in the renal connective tissue.

Case 7. *Small granular kidneys.*—James K—, stableman, æt. 54, was admitted to hospital February 17th last. He had suffered for six months from vomiting and indigestion, and from frontal headache. Eyelids and face had swollen in the morning recently. Eyesight had become dim. Some cough. Previous history good. No history of syphilis. When first seen the temperature was subnormal, and the pulse tension high. Heart's apex-beat in sixth space in nipple line. Soft systolic murmur at apex; second sound accentuated at base. Cardiac dulness absent. Scattered rhonchi over anterior chest; crepi-

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tation at the left pulmonary base behind. Edge of liver almost down to navel. Urine sp. gr. 1012, acid, albumen 0·12 per cent.; passed 24 oz. during first twenty-four hours. During his stay in the institution he had several short fits of unconciousness, muscles limp, no stertor or convulsions. The urine diminished in quantity as time progressed; once only 6 oz. were passed in twenty-four hours. Bleeding from the mucous membrane of mouth continued, with short intermissions, for the last ten days of life. About the same time lung symptoms became urgent. Crackling crepitant sounds, possibly due to friction, were heard over lower half of right lung. The day before death, on March 10th, the temperature rose to 100·1°, otherwise it was mostly subnormal. The autopsy revealed consolidation of the upper lobe of the right lung, its surface covered with shreds of lymph. On section a cavity, the size of a pigeon's egg, was discovered at the right apex, containing whitish friable material. The fibrous tissue was much increased throughout the lobe. Its texture was tough and airless; and its colour, a light grey, separated it sharply from the middle lobe. The rest of the lungs were œdematous and congested; some collapse at bases. Heart weighed 13 oz. Very flabby, pale, and dilated. Muscular fibres fatty. Both auricles much dilated. Tricuspid and mitral valves thickened and granular along their free edges. Aortic valve thickened, and a small vegetation on one cusp. Coronary arteries atheromatous. Just beyond the central cusp of aorta, and impinging on the orifice of coronary artery, was an atheromatous ulcer, the size of a shilling. It presented a fatty-looking base, with calcareous deposit just beneath the surface. The ulcer seriously obstructed the entrance of the artery. There was a second smaller ulcer about an inch onwards, and a number of calcareous plates, about the size of threepenny bits, for some inches along the arch. The kidneys were very small. Mottled pink and white. Capsules thickened. Surface pitted and granular. The granules were large and of a lighter colour than the

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intermediate portions. Numerous cysts were scattered over both organs.

Nuclear bodies were seen in the aortic adventitia. In kidney sections compressed tubules adjoined dilated ones. Many nuclear bodies were duplicate.

Case 8.—X. Y—, æt. about 40, a ticket collector on the Electric Railway, was brought to the hospital in a comatose state. He died shortly after admission. There was a history of a sudden fit. The brain was extensively lacerated by a large hæmorrhage into its substance. The arteries at the base were very atheromatous. Small patches of atheroma at commencement of aorta. Heart muscle hypertrophied. Valves thickened in parts, no growths along edges. Kidneys small and granular. Capsules adherent. Tissues tough.

Nuclear bodies can be seen scattered through the aortic coats, at the seat of an atheromatous patch. Some bodies are apparently about to escape from the inner surface of the intima into the blood-stream.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. vi, p. 25.)

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DESCRIPTION OF PLATE II.

A Disseminated Fibrosis of the Kidney found in cases of Infective Endocarditis, Aortitis, and allied disease (W. AINSLIE HOLLIS).

FIG. 1.—A portion of an aortic cusp, adjacent to a *corpus Arantii*, affected with atheroma circinatum, early stage. Case 6. Magnified about 5 diameters.

FIG. 2.—A portion of descending aorta, with atheromatous ulcer. The light parts were raised above the general surface of the endothelium. Case 7. Magnified about 4 diameters.

FIG. 3.—The upper part of the great aortic sinus, affected with ulceration due to atheroma circinatum. The endothelium was absent from the deeply-shaded portions of the ulcers, the edges of which were raised and everted. Case 6.

FIG. 4.—The adventitia of aorta with the openings of some vessels, showing the grouping of nuclear bodies around the vasa vasorum in atheroma. × 540.

FIG. 5.—Section of small mottled kidney, associated with multiple aortic ulceration. Case 7. A A, Tubules with epithelium swollen and granular. B, Malpighian tuft, with numerous nuclear bodies scattered over its surface. C C, Thickened interstitial tissue, with deeply-stained nuclear bodies within its meshes; a group of the latter at D. × about 600.

FIG. 6.—Section of Malpighian body in congestive mottling of kidney; early stage. Nuclear bodies, mostly apple-pip in shape, are grouped within the membranes of the tuft. Two or three are visible in the space between the tuft and capsule. Case 1. × 540.

FIG. 7.—Sections of atheromatous aortæ, showing nuclear bodies *in situ*. A A′, Inner surface of the aorta in Cases 1 and 8 respectively. At B a nuclear body is to be seen partially within the lumen of the vessel. In Case 1, that of a boy, the nuclear bodies are apparently more rounded in shape than they are in the adult, Case 8. The greater resiliency of the younger tissues possibly explains this difference of shape. × 540.

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Fig. 1.

Fig. 2.

Fig. 3.

Fig. 4.

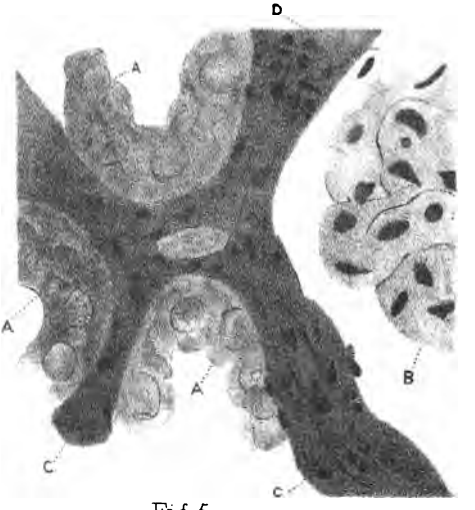

Fig. 5.

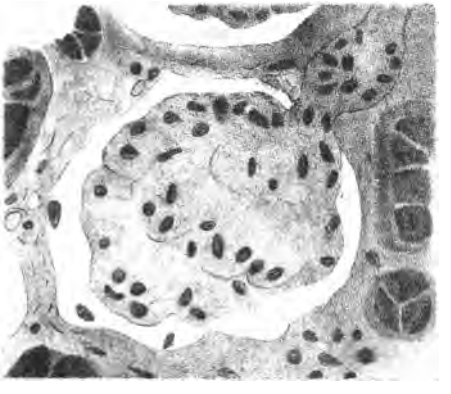

Fig. 6.

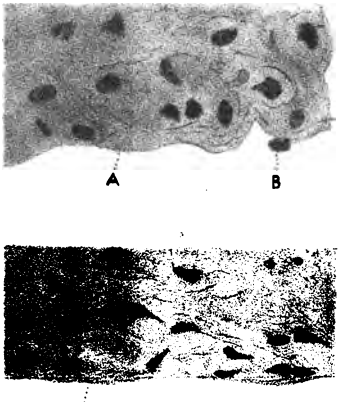

Fig. 7

Hollis & Lenanton, del.

Danielsson & Co, lith.

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ARTHRECTOMY OF ELBOW AND ANKLE.

BY

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Received July 5th—Read December 12th, 1893.

PREFACE.—My reason for bringing these cases before the notice of this Society is that surgeons do not as a rule, in the case of the elbow- and ankle-joints, follow the practice that they recommend and adopt for the hip and knee. I hope to be able to show that some modification of our treatment for these joints should be adopted under the improved circumstances of modern surgery.

ELBOW.

The old operation of excision of the knee has been much altered of recent years. The erasion or scraping away of all tubercular growth from every part of the joint is now the key-note of the operation. Whether a thin layer of cartilage is removed from the articular surfaces and the bones made to ankylose by pinning or wiring them together, depends upon the condition found at the operation and the views of the operator. As a rule, on account of the knee being in the centre of a limb where the chief function is that of supporting the weight of the body, it is better to aim at immediate

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ankylosis than to leave the knee to slowly undergo a subsequent contraction. If the cartilages are left, such rapid fusion of the two bones cannot be obtained.

In the case of the elbow, however, the same argument for removal of articular surfaces does not exist. The tubercular growths would by all modern surgeons be removed in the same thorough manner as in the case of the knee. This may be taken for granted, but the question which I wish to raise in this paper is, *whether it is necessary or desirable to remove the articular surfaces of the elbow-joint.* There are cases in which the operation of excision is rather too severe a method of treatment. Quite early in the disease no one would feel justified in taking off the ends of the bones forming the articulation. In young subjects, excision shortens the arm very materially, and the amount of movement to be obtained after the operation is very uncertain. Sometimes too much movement is obtained, and the new joint is flail-like in its action. Sometimes there is not enough movement produced for the amount of suffering experienced and trouble expended. Consequently the operation of excision is not done till quite late in the progress of the disease. The elbow in the early stage is therefore generally fixed at right angles on a splint in the hope that recovery will take place with ankylosis. Now it is in this stage that arthrectomy carefully done will shorten the duration of the treatment, improve the condition of the child's health, and often leave a movable articulation in place of an ankylosed one. If, however, the operation has not been undertaken early in the course of the disease, it must be an open question whether excision or arthrectomy would be the better operation. There is this to be said for arthrectomy, that even if ankylosis result the elbow is much stronger than after excision, the progress towards recovery is much more rapid and the operation is less severe, which is a point of some consideration in a weakly subject. Moreover, it is the result which you ordinarily expect to obtain by the treatment

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of rest on a splint without operation. So that the patient is not worse off than if recovery had taken place in the early stage by means of fixation on a splint, and all risk of a feeble or flail-like elbow is avoided.

But it is in the early stages of the disease that this operation is especially suitable. So soon as it has been demonstrated that the joint is not going to recover rapidly by the ordinary treatment of rest on a splint and good hygienic conditions, such as sea air and good food, &c., the operation of arthrectomy should, in my opinion, be recommended. The patient after operation at once improves in general health, and oftentimes to such a marked degree as to present quite a contrast to his or her previous condition. This is in part explained by the absence of pain, but who can doubt from the general experience of joint operations that the removal of the tubercular growth, provided suppuration does not take place, has a most marked effect upon the patient's general condition, even when the pain has not been a previous symptom of any importance.

If the operation of arthrectomy is done at this early stage, there is a very great probability that a movable articulation will be obtained *without any passive movement.* In none of my cases *was passive movement at any time employed.* Two of them became ankylosed, as the operawas undertaken at a late period of the disease, another suppurated and was eventually submitted to excision. But in the remainder the operation of arthrectomy was done fairly early, and a movable articulation was obtained without passive movement.

The operation of arthrectomy is conducted in the following manner, which is practically almost identical with that employed for the knee. The skin is thoroughly cleansed with soft soap or liquor potassæ to get rid of all the old epidermis. A transverse incision is made across the base of the olecranon, which is then divided with the saw. The olecranon is drawn upwards, and the fascial expansion on each side divided to still further permit its retraction. The joint

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is thus freely opened, and all parts can be efficiently scraped without further division of capsule or ligaments. With forceps and scissors, and the occasional use of a sharp spoon, all granulations are carefully removed.

Special attention is paid to all recesses such as that around the head of radius within the orbicular ligament. If the cartilage is loose it must be scraped away, and any soft and carious spot in the bone beneath must be gouged or scraped out. The most difficult part of the operation, however, is to clear the front of the joint beneath the brachialis anticus. If an assistant exercise traction on the arm and forearm the ligaments will be found to allow sufficient extension in their softened condition to enable the surgeon not only to introduce the sharp spoon between the articular surfaces but so effectually to scrape the front of the joint as to partially lay bare the muscle itself.

In most cases a finger even can be introduced and all cavities and recesses thoroughly explored. Whilst all this is being done, the joint is constantly flushed with warm boracic lotion, or weak perchloride of mercury solution, so as to wash out all the *débris* that accumulates from the scraping method of operation. When the joint has been freed from all the tubercular growth that can be detected, the olecranon is wired to the ulna in the same way that the patella is reunited in the case of the knee. The incision in the skin is completely closed with a continuous catgut suture, and a copious dressing applied to the back of the joint. No drainage-tube is now inserted, although one was employed for a few days in the earlier cases. A narrow strip of plaster of Paris is bandaged to the front of the arm, which is fixed at a right angle. In a favorable case this dressing is not removed for two or three weeks, when the wound is found healed and the catgut suture loose on the surface. If the temperature, however, should rise, the dressing has to be taken off for fear that there may be some distension of the joint. A director is in that case introduced between the edges of the incision and the fluid allowed to escape. When the

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wound has healed, the plaster-of-Paris splint is applied to the back of the joint instead of to the front, to protect it from any accidental injury. This is kept on for two or three months till all tenderness has disappeared. The patient is then allowed to use the arm in any way that he or she likes. *No passive movement* has at any time been employed in any of my cases.

This absence of any passive movement in the after-treatment of the case is of considerable importance. Which of us can forget our experience as house surgeons of the suffering that was endured by patients whose elbows had been excised? Daily passive movement continued over many weeks and sometimes months was then the rule of practice, and indeed is still considered necessary by those who excise the elbow-joint. But I question very much whether at the present time it is necessary in either variety of operation.

If the wound has followed an aseptic course, the movement will be very easily re-established by the patient's unaided efforts. If suppuration has accompanied the healing process, no amount of passive movement will do more than yield a very limited amount of mobility, and that same amount I would venture to state can be obtained without any forcible manipulation. In arthrectomy, if the cartilages are found firmly attached to bone, the movements voluntarily obtained are likely to be extremely good, in fact little less than of the normal range.

If the cartilages have already peeled off, the movements may not be quite so free, but the strength of the joint in pushing and traction will be far greater than after excision. In most cases, however, where the cartilages have gone the joint is likely to ankylose.

The nine cases which are here recorded represent my entire experience of this operation on the elbow-joint. Where excision has been the first operation it has not been included, but in the last case it will be seen that arthrectomy was followed by excision. The first two cases resulted in ankylosis for reasons which are suffi-

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ciently obvious in reading the history. But the disease was in each case arrested with the smallest amount of risk and discomfort to the patient.

CASE 1.—Edith J—, æt. 12, came from Wales and was admitted under my care at St. Thomas's Hospital in March, 1887. Her family history was bad. Her father and all her father's family had died of "consumption." She had lost one brother and one sister also of consumption. Three brothers and one sister were living. Her mother had died of heart disease.

Previous history.—At six years of age her left thigh had been amputated for disease of the knee.

The *present trouble* in the right elbow had begun five years before by its being painful and weak. During the last month or two the joint had been much more painful, and kept her awake at night.

There were the ordinary signs of disease with tenderness along the line of articulation, but no sinuses. The urine contained a trace of albumen. She was an extremely delicate-looking girl, but had no obvious signs of phthisis.

April 2nd.—The operation previously described was performed. There was a large amount of gelatinous synovial membrane to remove from all the recesses of the joint. The cartilage peeled off in large flakes from the bone, which was found soft, crumbling, and in parts replaced by tubercular granulation tissue. There were neither sequestra nor distinct cavities in the bone containing caseous *débris* of the tubercular growth.

The operation wound healed at once and required only a few dressings. Normal temperature was maintained after the first two days, and at the end of May she left for a convalescent home with the elbow in a plaster-of-Paris splint. There were then three very small tubercular sinuses which produced scarcely any discharge.

July 27th.—She returned to hospital with the sinuses healed. She had grown quite fat and strong, her appearance being in marked contrast to her previous condition.

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The joint was ankylosed firmly at right angles, but she could use it freely and suffered very little inconvenience from the ankylosis. She had arrived more rapidly at the best result that would follow from the fixation of a diseased elbow in a splint than she would have done without any operation. She might, it is true, by excision have obtained a movable joint, but a glance at her history and physical condition did not lead me to expect a good result from excision. In her case I was more than satisfied with having arrested the progress of the disease and obliterated the joint as a nidus for further trouble.

Case 2.—Elizabeth S—, æt. 12, first came under my notice at St. Thomas's Hospital in March, 1884, for disease of right elbow after a fall a few weeks before. The elbow was put up in a plaster-of-Paris splint, and she ceased attending in July, 1884.

In April, 1887, she again came under observation complaining in the same way of pain and swelling in the right elbow, after a fall about Christmas time. She was now unable to sleep on account of pain. The limb was fixed in plaster of Paris.

In September, 1887, she was admitted into hospital, as, although she was decidedly better after the four months' rest, the joint still presented the ordinary signs of active tubercular disease.

There was one point of importance in the family history, namely, that her mother had died of phthisis; she had had one sister, who died when a young woman from some cause which was unknown.

The right elbow on examination still proved to be slightly movable on flexion and extension. There was a good deal of swelling all round the joint, especially over the head of radius, and in parts it was distinctly fluctuating. There was lateral mobility and extreme tenderness on pressure all along the line of articulation, but there was no sinus.

September 17th, 1887.—The joint was opened in the

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manner already described, and a quantity of purulent-looking fluid immediately escaped. The tubercular synovial membrane was all removed with sharp spoon and scissors, especially from the front of the joint and around the radio-ulnar articulation. The cartilage had almost entirely disappeared, and the bone beneath crumbled away before the gentlest application of the spoon. Nearly the whole of the head of the radius and a large portion of the external condyle of humerus were removed in this way. After the first five days the temperature remained normal, and the splint was removed on October 18th. It was then free from swelling and pain, and the wound was soundly healed.

She was sent to a convalescent home on February 7th, 1888, in excellent health. The joint was free from swelling and tenderness and firmly ankylosed, but there was one tubercular sinus.

April, 1888.—The wound was perfectly healed, and there was no sign of disease; but as there was no chance of obtaining movement a leather splint was ordered to protect the joint from any accidental injury.

January, 1889.—She was fatter and stronger than I had ever seen her before. The joint was perfectly ankylosed in good position, and free from any sign of disease.

Case 3.—Eleanor T—, æt. 5, was admitted into the Victoria Hospital for Children under my care in March, 1888. "Family history of phthisis on mother's side." There were no other children.

There was a vague account of a fall on the elbow one and a half years before, and of a painless swelling for twelve months. A gutta-percha splint had been applied, and at the end of six months an abscess was opened. On admission there was much swelling of the left elbow-joint, with two sinuses, one on each side of the articulation.

March 26th.—The joint was opened by a transverse incision and division of the olecranon. A great deal of tubercular synovial membrane was removed, and carious

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bone in the olecranon fossa was cleared away with the gouge. The cartilage was unaffected except at the spot just mentioned and at the margin of the articulation. I had some hope that this would in the end prove to be a movable joint, but suppuration took place and the temperature rose from time to time; the septic changes were, I think, due to the presence of sinuses before operation. She went to Margate on June 6th, and was last seen on October 29th, 1888. The joint was then healed, and there was a little movement. But she has been lost sight of, and there are not sufficient notes to speak positively of the final result, except that the disease was at an end.

Case 4.—Ethel H—, æt. 6, was admitted into the Victoria Hospital for Children on October 22nd, 1888. There is no record of her family history. The disease was attributed to a fall on her elbow six months before, as the injury was immediately followed by swelling, pain, and stiffness. She had been treated by a splint. On admission there was considerable swelling of left elbow-joint with distinct fluctuation above the olecranon. Flexion and extension were extremely limited, but pronation and supination were unimpaired.

November 12th.—The joint was opened by division of olecranon, and a large caseous cavity was found in the lower end of the humerus extending into the trochlea and making a communication between the coronoid and olecranon fossæ. A large quantity of tubercular synovial membrane was removed from the front of the joint and from beneath the orbicular ligament. The cartilage and bone were healthy except at the spot above mentioned. The joint healed by first intention and gave no trouble or anxiety. Temperature rose to 100° within the first four days, but was afterwards normal. She went to Margate on December 6th with a fair amount of movement in her elbow. She had no passive movement of any kind, no recurrence of disease took place, and she has slowly acquired a movable joint. Flexion and exten-

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sion are quite as free as a patient obtains after excision with the best results; and, moreover, the movement is in the normal plane, *i. e.* without any lateral deviation. The strength and power in extension of the elbow-joint is far beyond anything that is seen after excision. Pronation and supination are scarcely less free than in a normal joint.

She was shown at the Medical Society in February, 1892, and these points demonstrated, and at the Royal Medical and Chirurgical Society, December 12th, 1893.

Case 5.—Charlotte F—, æt. 8, was admitted into the Victoria Hospital on October 3rd, 1888. There was no history in her family of tuberculosis, and no definite account of injury. Pain and swelling in right elbow were first noticed in May—five months before her admission. As she had had no treatment by rest, the elbow was placed in a plaster-of-Paris splint and she was sent to the Margate Convalescent Home attached to the hospital on October 24th.

November 21st.—She was readmitted into hospital. There was no improvement visible in the condition of the joint. There was very considerable swelling of the elbow with rigidity and an unnatural lateral mobility showing that the ligaments were softened.

December 3rd.—The joint was opened by transverse section of the olecranon and a very extensive tubercular growth from synovial membrane exposed. This was all removed by scalpel, scissors, and scraper. The cartilage over the coronoid process came away, but in other parts it was firmly attached to the bone beneath.

By December 10th the wound was healed. There had been no pyrexia. On the 19th she was discharged.

May 6th, 1889.—She was readmitted with recurrence of disease in the external condyle of humerus. On May 13th the second operation was performed. This consisted in making a straight incision over the external condyle and the head of the radius; a good deal of bone was scraped away, and the cartilage from the capitellum and the head of the radius was removed. On May 22nd the wound was

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healed, and she was discharged with a splint. There had been no pyrexia.

August 11th, 1890.—She was readmitted with a small discharging sinus over the external condyle which had existed for a few months.

25th.—Third operation. The sinus was enlarged and a cavity in the bone scraped. There was no fever, but the sinus did not heal. She was therefore sent to Margate on September 17th.

After each operation this child wore a splint for two to three months, and passive movement was not at any time employed. She finally obtained complete flexion of the elbow-joint, and could extend the forearm about halfway beyond the right angle, towards complete extension. She had therefore a very wide range of movement in flexion and extension. Pronation and supination were, however, limited to half the normal range. She was shown at the Medical Society in February, 1892, in the condition described above.

Case 6.—Bessie F—, æt. 9½, was admitted into the Victoria Hospital on March 15th, 1888. There was a family history of phthisis on both the father's and mother's side, but no history of any injury.

Ten months before admission she had first complained of pain in right elbow. The joint became swollen and stiff, but no treatment by rest had been adopted. On admission there was little or no movement in the joint, and a large amount of synovial thickening with fluctuation on either side of the olecranon. Although she had had no splint applied, I considered it more probable that she would obtain a movable joint if the diseased tissues were first removed by arthrectomy than if the joint were simply kept at rest.

April 30th.—The joint was opened in the same manner as in the other cases. The front of the joint and the head of the radius were comparatively free, and the main bulk of the disease was situated in the posterior part of

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the joint. This appeared to have started from a cavity in the ulna between the olecranon and coronoid processes. This was scraped out and all the tubercular synovial membrane removed. The wound was healed on May 17th and she was discharged on May 29th.

No passive movement was at any time employed, and a splint was worn only for a short time. She came to report herself from time to time, and was shown at the Medical Society in February, 1892.

She has as perfect power of flexion as would be seen in a normal joint. But extension can only be obtained by the patient to a point a little beyond the right angle, whilst pronation and supination are quite as good as in a healthy joint.

She was shown to the Society December 12th, 1893.

CASE 7. *Disease of right elbow, left wrist, and caries of cervical spine with paraplegia.*—Louis W—, a boy æt. 9, was admitted into the Victoria Hospital in June, 1890, with disease of right elbow-joint which was most marked in the radio-ulnar articulation. He was sent to Margate with the elbow in a plaster-of-Paris splint on July 2nd, and was readmitted on October 27th with the joint practically in the same condition as before. On November 3rd the radio-ulnar and humeral joint was exposed. A cavity in the ulna was scraped out and much granulation tissue was removed from the joint around the head of radius. On December 8th he was discharged from the hospital with the wound healed and a fair amount of pronation and supination. A posterior plaster-of-Paris splint was applied to protect the joint from injury.

May 7th, 1891.—He was readmitted with well-marked recurrent disease in the whole of right elbow-joint. His left wrist, which had in the previous year been swollen and tender and consequently treated by fixation on a splint, was now decidedly diseased.

May 25th.—The right elbow-joint was opened by transverse division of the olecranon and the diseased

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tissues scraped away. Very little of the head of the radius was left at the end of the operation. The wound healed rapidly as before, and on June 11th the left wrist was partially excised. The joint was opened by a single straight incision, and those of the carpal bones which were soft and loose were removed and the granulation tissue scraped away. This wound also quickly healed, and he left the hospital on June 27th.

In August he was seen at home by Mr. Nairn, who was then acting as house surgeon to the Victoria Hospital, and found to be suffering from cervical caries with paralysis of both arms, legs, and intercostal muscles. It was not at that time thought safe to move him to the hospital. He improved a little during the autumn, and was readmitted into hospital in February, 1892. He still had almost complete paralysis of arms and legs, but the intercostals were acting feebly. He had also a retro-pharyngeal abscess, which was opened externally behind the right sterno-mastoid. He developed under observation a swelling of the right half of the lower jaw, which proved to be due to caries. The two joints which I had previously operated on were free from gross disease, but the scar over the elbow was not quite sound. There was a little movement in the elbow-joint, and the position was good. The wrist-joint was very movable, and the fingers as pliable as those of the other hand.

He slowly recovered from the paralysis, and left the hospital in January, 1893, but the sinuses in the neck and under the jaw were still discharging. He died of tubercular meningitis in May, 1893.

Case 8.—Lillie W—, æt. 4, was admitted into St. Thomas's Hospital on January 21st, 1891, with a history of only seven weeks' trouble in the right elbow after measles. The right elbow was very large and fluctuating all over, and thought to be filled with pus or softening tubercle. Temperature normal. It was not tender on examination, and movement could be obtained without

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making the patient cry. Lateral mobility was very marked and rather painful. On the whole it was thought most likely to be a case of tubercular synovial disease, on account of the absence of acute pain and tenderness and the normal temperature. Chloroform was given on January 27th, and an incision made through the triceps into the centre of the swollen joint. Turbid fluid escaped, but the swelling was not much diminished. The wound was closed with a continuous suture, dressed, and placed in a plaster-of-Paris splint. No symptoms whatever followed this exploration, and the temperature remained normal.

March 7th.—The outer side of the joint was now much more swollen than any other part, and distinctly fluctuating. Chloroform was given, and an incision was made into the outer side of the joint. Pus escaped, and a finger being introduced a cavity was found in the outer condyle of the humerus containing a small sequestrum. This was removed, and the cavity scraped out with a sharp spoon. The cartilage was firmly attached over the whole of the articulation which was within reach. The joint was washed out and a drainage-tube introduced for a few days, and the arm fixed as before in a plaster-of-Paris splint.

April 17th.—The wound has been healed for some time. There is no swelling of the joint and no sign of disease. Some movement can be obtained without pain.

May 13th.—The limb was rather too straight from want of the splint, which had been left off without sanction. Plaster of Paris was reapplied with the elbow in over-flexed position.

After this the child was lost sight of for many months. The mother was written to, but no answer was received and the letter was not returned.

May 18th, 1892.—Twelve months after last note the child reappeared on account of tubercular dactylitis, for which a splint was ordered. The elbow was then found to be in a most satisfactory condition. There was no sign of disease. Flexion could be voluntarily obtained to half

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a right angle, whilst the power of extension was nearly normal. Pronation and supination were perfect.

CASE 9.—Herbert A—, æt. 21, first came under my observation in consultation with Dr. Hamilton Allen, in February, 1890. He had complained of weakness in the right elbow since the previous November. A swelling had formed about a month before his visit, and had been opened with considerable relief by Dr. Allen. He had a sinus just below the inner condyle of right elbow, the probe passing deeply to the ulna but not absolutely striking dead bone. The joint was apparently free from any sign of disease.

April 19th, 1890.—This sinus was enlarged and the finger was introduced. The ulna just below and behind the seat of articulation was found rough and carious. This surface was scraped with a sharp spoon and the joint was fixed in plaster of Paris. The case did well till August, when it was found that the joint had become involved, with swelling, much pain, and starting at night.

August 29th, 1890.—A complete arthrectomy by transverse division of olecranon was performed in St. Thomas's Home. The operation was a complete failure. Sepsis undoubtedly took place, with profuse discharge and high temperature.

In October he went to Margate, and returned in the middle of November with well-marked disease of elbow and several sinuses. By the kindness of Mr. Watson Cheyne he underwent Koch's treatment in King's College Hospital during December and January, but with no beneficial result.

April, 1891.—He was admitted into St. Thomas's Hospital with sinuses on both sides and in front of the elbow-joint. There was a great deal of discharge and swelling, but no pain.

May 2nd, 1891.—The elbow-joint was excised, by making an H-shaped incision, on account of the sinuses. A small fragment of the olecranon was saved to attach to the ulna for the sake of the triceps, but in all other

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respects the operation resembled the old-fashioned excision. There was a good deal of subarticular caries, but the bone beyond this area was healthy. All the gelatinous synovial growth, which was very profuse, was removed, the operation on this account lasting an hour and a half.

This operation was satisfactory, for in June he was attending as an out-patient and being dressed twice a week. There was then very little discharge, and the elbow had contracted to a moderate size.

December, 1891.—The sinuses still existed, but there was a limited amount of movement and very little discharge.

September 22nd, 1892.—The sinuses exist, but the discharge is insignificant; there is no pain and some movement in the joint.

June 23rd, 1893.—Dr. Allen sent this patient to me again on account of a chronic lumbar abscess. His elbow was almost well. There was no swelling and a fair amount of movement. There were still a few superficial spots with occasional discharge. It was a very useful elbow for all ordinary purposes.

Ankle.

Nearly all surgical writers unite in condemning excision of the ankle-joint, and state that a Syme's amputation is far more satisfactory for those cases which do not recover by rest on a splint.

Since 1887 every ankle-joint which has required operative interference at my hands has in young subjects been submitted to arthrectomy by multiple incisions. One case only, in a man aged sixty-nine, was submitted to amputation.

The six cases here recorded of this operation are sufficiently encouraging to make one think that more effort should be made in the conservative treatment of this joint than appears at present to be the practice of operating surgeons. It must, however, be of limited application, for many cases in which the ankle-joint is involved have well-marked disease of nearly all the tarsal bones.

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Obviously this operation would be unsuitable if both the whole tarsus and ankle-joint were involved. It is not my intention to speak here of disease of the tarsus, but of the ankle-joint proper.

In one case, in addition to the ankle-joint, the os calcis was extensively diseased and required almost complete removal from its periosteal shell. But with this exception the cases were those of true ankle-joint disease, in which, however, more or less of the astragalus was gouged away. They were at first treated by plaster-of-Paris splint and a knee-rest, unless they were too young to use this means of locomotion. It was only after this had failed to produce a good result that the operation was performed.

The method of procedure was by a series of vertical incisions round the ankle-joint, through which a sharp spoon and even a finger could be introduced. Four incisions, one in front and another behind each malleolus, avoiding ligaments and tendons but freely opening the joint, were generally employed. If traction was made on the foot, the finger could always be introduced to examine the joint surfaces after the use of the sharp spoon. The nozzle of an irrigator with a full stream of aseptic fluid was kept constantly going through one or other of the openings round the joint. The introduction of the finger was especially useful in hunting for soft patches of tubercular granulation tissue, for then the sharp spoon could be directed to that spot. In most cases the cartilage came away and the bone beneath was attacked with more or less vigour, according to the condition of the fragments which were removed. An objection has been raised to this method of procedure, namely, that the surgeon cannot *see* the condition of the structures upon which he is operating. But if the finger is used as described above, and when the bone is being scraped the fragments are carefully examined, there is really little difficulty in arriving at a conclusion as to when enough has been done. The consistence of the bone and its resistance to the action of a sharp spoon is not alone suffi-

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cient, for, in the neighbourhood of a diseased articulation, the bone is often rarefied without being invaded with tubercle. When the operation was finished, a drainage-tube was usually inserted into the two posterior openings and retained for a few days, but latterly this has been given up. A copious dressing was applied, and over this a plaster-of-Paris splint was put on with an iron bar at the back, and windows at each side to enable us to watch for oozing of blood and discharge. When the patient was able to get up, a knee-rest was ordered to be used for many months.

Brüns has described exactly the same method of operating in the 'Münchener med. Wochenschrift,' 1891. It is, in fact, the plan which any surgeon would adopt who wished to scrape out the ankle-joint without a very free division of the tendons and ligaments round this articulation.

Mr. Arbuthnot Lane has recently (November, 1892) described at the Clinical Society two cases in which he opened the ankle-joint for the removal of disease by an incision across the front and outer side of the articulation, dividing all the tendons except those behind the internal malleolus. After the disease had been removed, he reunited the tendons and obtained an excellent result so far as the disease was concerned. Possibly this may be the better way of thoroughly clearing the joint of all tubercular growth, but it is a severe measure of which I have not as yet made a trial. After the results that have been obtained by the method described in this paper, I should feel inclined to think that Mr. Lane's plan should be reserved for cases of very advanced disease in which the growth has extended beyond the capsule of the joint.

But in the ankle, as in other joints, an early operation on the lines here indicated would I think save most cases from such extensive operative measures.

Mr. Lane could not give the precise results in his first case as to power of walking and mobility of the toes.

In the second the child could not move the toes, and the tendons had since required shortening. Information

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is still wanting as to the result of this secondary operation and on the usefulness of the foot in walking. Both were cases of such advanced disease that a Syme's amputation was the only alternative. So far this is satisfactory, but it is necessary to show that the power of walking is as good as it would be after amputation to gain converts to this extensive division of the tendons of the ankle-joint.

Case 1.—Charles R—, æt. 9, was sent to me at St. Thomas's Hospital by Dr. Weddall Thomas, of Peckham, in November, 1886, with the following history. Six years before, at the age of three, he had received an injury to his right ankle. This was followed by an abscess, which healed in a few months. He remained well for three years, when he was again lame for a short time. Two months before I first saw him was the date of the commencement of the attack for which he sought advice. The right ankle was swollen and very tender. There was a cicatrix close to the external malleolus. He could walk, but was very lame. The ankle was placed in a plaster-of-Paris splint and a knee-rest was ordered. He was given cod-liver oil and iron.

March, 1887.—The ankle was very little if at all improved; he had really carried out the principle of perfect rest to the joint for five months. He was therefore admitted into hospital for operation. There was gelatinous synovial thickening all round the articulation, filling up the hollows and depressions, but most marked in the neighbourhood of the external malleolus where the scar was placed. Here also there was more tenderness on pressure than elsewhere. Passive movements of the joint were comparatively free and unaccompanied by pain.

The operation previously described was performed, by four vertical incisions, one in front and another behind each malleolus. About an ounce of purulent-looking fluid was evacuated and a large quantity of gelatinous synovial membrane was removed with the sharp spoon from the whole articulation. The finger was easily introduced, and

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a cavity was found in the external malleolus communicating with the tibio-fibular portion of the common joint cavity, This was thoroughly scraped out and the softened bone around removed. The scar previously mentioned was exactly over this cavity, which was no doubt the spot at which the disease had originated in the lower epiphysis of the fibula and from which the whole joint had become infected. The temperature after the operation never rose above 99°, and the wound healed rapidly, leaving, however, small sinuses with scarcely any discharge for a few months. He went to a convalescent home in June with his leg on a knee-rest, and returned in August. His ankle was then in first-rate condition without swelling, pain, or tenderness, but there was still one sinus over the external malleolus.

February, 1888.—This sinus leading to the tip of external malleolus was scraped, and then it finally healed. He still continued both splint and knee-rest, although there was no sign of disease in the ankle-joint. In November, 1889, he was allowed to walk about at home but still to use the knee-rest out of doors. The joint was then quite movable and free from disease.

December, 1890.—For some time it had been noticed that, although the ankle was free from disease, the foot was becoming everted from irregular growth at the epiphysial line of the tibia, so that the centre of gravity did not fall upon the tarsal arch but to the inner side. Apparently this was due to deficient growth on the outer side of the tibial epiphysis, for the fibula was bent and the internal malleolus unduly prominent; but it might have been due to overgrowth at the inner side of the epiphysial line.

In February, 1891, an osteotomy of the tibia and the fibula was done about 1½ inches from the joint line, removing a little bone from the inner side of the tibia in the form of a wedge. The foot was then easily brought into the correct position and fixed in plaster of Paris. The wounds healed at once and the deformity was corrected.

In June he was walking about with boot and side-irons to maintain this improved position.

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October, 1892.—For the past twelve months he had been acting as a hosier's assistant, standing for twelve hours behind the counter lifting heavy boxes and parcels, &c., from the shelves. He was not lame, his ankle was movable to a slight extent and free from disease. To see him walk no one would suspect that he had had disease of his ankle. He came only to ask if he ought to renew the double irons that were supplied in June, 1891, as he had outgrown them.

He was shown to the Society December 12th, 1893.

Case 2.—Réné van der V—, æt. 3, was sent to me at St. Thomas's Hospital by Dr. Cook, who was at that time in practice in Honor Oak. His ankle had been swollen and painful for four months, and a discharging sinus had existed for six weeks. There was a history of a blow two months before the ankle was first noticed to be swollen. On his mother's side the grandfather had died of phthisis, but there was no other history of consumption, and his mother had had three other children, who were alive and well. The right ankle was much swollen with tubercular synovial membrane all round the articulation. Above the internal malleolus there was a large open sinus leading directly into the tibia at the epiphysial line. The child was admitted into hospital, and on August 13th was submitted to operation. The sinus was enlarged and the bone scraped. A carious cavity was found at the epiphysial line, formed chiefly at the expense of the shaft, from which the epiphysis was almost detached and loose. This was on the wrong side for the disease to have spread to the ankle-joint by continuity. Still the path may have been present but unobserved. The ankle was opened by four incisions as before described, and all the granulation tissue, of which there was a great quantity, was scraped away. The cartilage on both the tibia and the astragalus was seen and felt to be firmly attached to the bone beneath. The highest temperature recorded was 100° F., and the sinuses had almost healed when in October

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he was found to have contracted scarlet fever. This he had in rather a severe form, followed by albuminuria. In February, 1888, he came back to my charge in a feeble condition. But the joint, although discharging more than it had done when he left the surgical ward, was not in a hopeless state. There was not much swelling, very little pain, and the ligaments were still intact. The friends, however, were convinced that he was going to die, and insisted on removing him from the hospital. They had not seen him whilst he was suffering from scarlet fever, and judged by the altered appearance of his face that the ankle was the cause of his having become thin and weak. To my mind there was a very good prospect of perfect recovery both of health and of joint, but the case was not seen again and no positive statement can be made of the result.

Case 3.—Frederick Walter L—, æt. 7, was admitted under my care at the Victoria Hospital for Children in May, 1889. He had had measles a year before, and shortly after an injury to his left ankle, which had never been well since.

There was a great deal of pulpy swelling all round the line of articulation, with tenderness on pressure and pain on movement. He had been treated as an out-patient by rest, but without any improvement. He was therefore, after a few weeks' confinement to bed, submitted to arthrectomy of the left ankle. The same method of operating was employed, and the tubercular granulation tissue was scraped away. The cartilage over the tibia and the astragalus was loose, and came off in large flakes. A good deal of the astragalus had to be gouged away before sound bone was reached. He was sent to Margate in June with his leg on a knee-rest. He was readmitted in August with sinuses, but his general health was very much improved. The sinuses were scraped, but no disease of bone was discovered. The wounds were all soundly and permanently healed by the end of September, and he left the

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hospital in October, 1889, with his leg still on a knee-rest. He was made to continue the use of the knee-rest during the whole of 1890, but in 1891 he was allowed to give it up whilst indoors.

In 1892 he was allowed to walk with boot and iron, as he had had no relapse and the joint had remained free from any sign of disease.

On October 20th, 1892, the following note was made:—"The joint is ankylosed at right angles, but there is free movement in the mid-tarsal joint, so that there is scarcely any perceptible lameness in walking."

He was shown to the Society December 12th, 1893.

Case 4.—Nellie K—, æt. 2, was admitted under my care into the Victoria Hospital for Children in August, 1889. She had well-marked disease of the ankle and inner side of tarsus, which had commenced about three months before admission. The foot was put up in plaster of Paris, and the child sent to the Convalescent Home at Margate attached to this hospital. She was readmitted at the end of five weeks, when the disease was found to have steadily progressed in spite of the enforced rest and good hygienic surroundings. An abscess was at once opened on the inner side of foot, and on September 25th a complete arthrectomy was performed. The same method was employed as in the other cases, but the disease was much more extensive, involving the astragalus, os calcis, and scaphoid, besides the whole of the ankle-joint proper. These bones had therefore to be scraped and gouged in a way that did not give much prospect of success. But so far as the immediate result was concerned it looked most hopeful, for the wounds rapidly healed. At the end of October recurrence of disease was, however, clearly established, and a second scraping operation was performed. Again the wounds did well for a time, but in December a third operation of the same kind became necessary. On this occasion nearly the whole of the os calcis was removed from within by the gouge, leaving only a shell of bone. A good deal of the articular end

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of the tibia had also to be removed. She left for the Convalescent Home at Margate on January 15th, 1890, about five weeks after this last operation, with, as I thought, poor prospects of a useful foot. However, it recovered from these severe operations in the most marvellous manner, and by June, 1890, the wounds were healed and the foot was free from disease. I certainly did not expect such a good result, but as the child was healthy and bore these operations well without any marked constitutional disturbance or septic contamination, I thought it worth while to persevere, as the foot could at any time be removed by Syme's amputation.

The child has unfortunately not been seen since June, 1890, and letters sent to the former address have been returned through the post. If the child had been old enough for a knee-rest, she would undoubtedly have been brought back to know if it might be left off. As it was the child walked about in a plaster-of-Paris splint, and this soon ends in the splint being removed by the mother, and if there are no wounds to dress and no pain, she does not think it necessary to come to hospital. We may therefore conclude that the case has been successful.

Case 5.—Lydia W—, æt. 6, was admitted into the Victoria Hospital under my care in September, 1890. Her right ankle had been swollen and painful since December, 1889. She had worn a plaster-of-Paris splint and used crutches since March, with considerable benefit. At the time of her admission the ankle was steadily getting worse, with increasing swelling, more pain and tenderness, and "night starts."

On October 13th, 1890, complete arthrectomy of the ankle-joint was performed with the same incisions as in the previous cases. The synovial membrane was freely removed and the cartilage easily peeled off from tibia and astragalus.

It was found necessary to remove a good deal of the astragalus with spoon and gouge, but the tibia seemed fairly firm and sound. The temperature remained normal,

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and the wounds were healed by January, 1891. She left for Margate on January 15th with her leg on a knee-rest and her foot in plaster of Paris.

There has been no relapse, and the ankle is now perfectly well. She began walking in May, 1892, with boot and iron, and has been seen many times since then. At the present time (June, 1893) she walks without lameness, has a fair amount of movement in the ankle-joint, and looks the picture of health.

She was shown to the Society December 12th, 1893.

Case 6.—Florence B—, æt. 6, was admitted under my care into St. Thomas's Hospital in August, 1890. Eight months before admission she had slipped off the kerbstone and injured her left ankle, which was swollen immediately after and had never properly recovered.

There was no family or previous history of importance. She had been treated by me in the out-patient room for six months with plaster of Paris and a knee-rest, but no marked improvement had taken place. There was swelling all round the left ankle of the kind usually seen in tubercular arthritis. There was distinct tenderness along the line of articulation, but there was still some movement in the joint. Arthrectomy was done in September in the manner before described. Pus was found towards the outer side of the joint. The cartilage came away from both the tibia and the astragalus, and a large part of the latter was removed, as it was thought from examination of the fragments that it was tubercular.

Early in October the wounds had healed, the joint was movable, and there was no pain or tenderness. She was sent to a Convalescent Home with her leg on a knee-rest.

June 19th, 1891.—She was readmitted. She had not been seen since the preceding October, but had worn her knee-rest ever since, and had improved immensely in general health. There was a painless gelatinous swelling all round the articulation, but the tarsal bones and joints were quite free from any signs of disease. In July the

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joint was opened in the same manner as before, but with the exception of a small amount of tubercular growth beneath one of the original wounds there was no disease to be detected. The joint surfaces looked and felt quite healthy. The wounds healed rapidly and she became an out-patient, coming up from time to time for examination. During the whole of 1892 she continued to use a knee-rest and a plaster-of-Paris splint with an iron bar at the back. No recurrence of disease took place, although from the polished condition of the iron bar round the heel and sole of foot it was clear that she walked about at home.

January, 1893.—She was ordered a boot and iron for walking. There was no sign of disease, and some movement was retained in the ankle-joint. She has been seen several times since this note was made, walking well but with some stiffness in the ankle-joint. Increased mobility, chiefly in the tarsal joint, will take place as in the other cases with time and exercise.

April 12th, 1893.—The movement in the ankle-joint is decidedly increasing.

Shown to the Society December 12th, 1893.

Two other cases might also be mentioned in relation to this operation, although they have not been included in the above list because the whole ankle-joint was not submitted to arthrectomy.

Both were cases in which the head and neck of astragalus were partially removed by scraping and gouging. The ankle-joint was opened through the incision necessary for this operation, and the anterior part only of this joint was treated with the sharp spoon. In one of these cases complete recovery took place, and the child is now walking about with a sound foot. In the other the disease returned, and during my absence from work last summer (1892) was submitted to amputation. I am unable therefore to say what was the condition of affairs.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. vi, p. 29.)

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ON ALBUMINURIC ULCERATION OF THE BOWELS.

BY

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I PRESUME to bring before the Society an accompaniment and result of renal disease to which I have formerly briefly referred, but which has not yet received the notice which is due to it, whether as regards its pathological interest or its vital importance. I am not aware that the concurrence of ulceration of the bowel with albuminuria found mention until 1876, when, in the Croonian Lectures of that year, I ventured to adduce two cases of this enteric lesion in connection with the granular kidney, unexplained by either typhoid or tubercle and not to be accounted for otherwise than as related in some manner to the renal disease.[1] The late Dr. Greenhow presently enabled me to add a third, which, with the two before mentioned, was cited in my second edition of 'Albuminuria' in 1877, with the recognition that the intestinal change resulted from the renal,

[1] 'British Medical Journal,' April 22nd, 1876, p. 560.

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though at that time it was not apparent how the association was brought about. In the following year I called the notice of the Pathological Society to a case of "Ulceration of the Bowel as a Consequence of Renal Disease,"[1] with the particulars of a microscopic examination of the bowel, made by my former clinical clerk Mr. A. Shann, which revealed the hæmorrhagic nature of the process.

During the considerable time which has passed since these observations were published I have been on the watch for the enteric complication of albuminuria, which has long taken its place—at St. George's at least—among the things to be looked for in post-mortems relating to renal disease. I now propose to ask the attention of the Society to the experience which has accumulated, making use not only of my own cases, but also of those which have occurred in the practice of my colleagues, to whom I tender my thanks for their permission to do so. To avoid the length and tediousness of complicated narratives, I have condensed the records into a tabular form, which will present in brief the essential particulars. These have been for the most part gathered from the hospital post-mortem books, though I have occasionally been able to refer to my own notes in addition. The observations extend over forty years. The first case came under my notice when I was clerk to the late Dr. Bence Jones, though the connection of the ulceration with the renal disease was not recognised until later instances had thrown a backward light upon it, and enabled it to be recovered from a remote past to illustrate comparatively recent pathology.

I have brought together twenty-two cases of ulceration of the bowel, concurrent with albuminuria and not with any other condition by which the enteric lesion could be explained. In two of them there was also ulceration of the stomach, possibly of the same nature, and a single instance will be found concerning the same organ which must be ad-

[1] 'Path. Soc. Trans.,' vol. xxix, p. 117.

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mitted to be of doubtful value, though of some interest in relation to the conditions under which it occurred. I have appended, with design to throw light upon the morbid process, eight instances of extravasation of blood without ulceration, but similar in place and circumstances to it.

With regard to the bowel, the ulceration was confined to the small intestine in eleven cases; confined to the large intestine in five cases; in five both were affected. In one the position of the ulceration was not more exactly defined than as "near the cæcum." Within the small bowel the region most frequently affected was the ileum, though no part was exempt. In the large bowel the colon suffered with the greatest frequency, though the cæcum was affected alone or with other parts of the bowel in three instances.

The ulcers were not associated in particular with any of the glandular structures of the intestines, but affected the mucous membrane without any obvious preference excepting in general terms for the lower part of the ileum. Peyer's patches were usually exempt, though in one instance (that of Williams) they were involved. The solitary glands of the cæcum were recorded in one case as intensely congested but not ulcerated. In several the valvulæ conniventes were found to be either ulcerated or congested, so that if the morbid process have any special choice, however rarely exercised, it would seem to be for these structures. In size the ulcers varied from two inches by one to spots no larger than the marks of raindrops. The ulcers were usually regular in shape, more or less circular, without thickening, and with "punched" edges. In some instances the ulcerated surface had become covered, as if by a process of healing, with a delicate membranous film (Jackson, Hissgen).

The most conspicuous characters of the morbid change were derived from hæmorrhage, old and new, in the neighbourhood of the ulcers. Pigmented stains and recent ecchymoses were common—the latter in situations, such as the edges of the valvulæ conniventes, where the

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ulcerative process has been known to declare itself. The most important characteristic of the ulcers is their tendency to perforate and set up peritonitis, in other words their deep origin or penetrating character.

This introduces the minute anatomy of the ulcers and the morbid process to which they are due. The most constant factor, and that which presents itself as of the greatest significance, is extravasation of blood. The earlier observations which led to this conclusion have been corroborated by more recent; I may refer especially to some microscopic examinations which Mr. Fenton has recently undertaken at my request. These relate to two preparations in our museum which had not hitherto been examined in section. They belonged to the cases of Hissgen and Parker; and though they had been for fourteen years in spirit, there was no difficulty in recognising the hæmorrhage connected with the ulcers. It is probable that the effusion of blood in the submucous coat, which has been microscopically demonstrated in three instances, is the primary and essential lesion upon which the ulceration is consequent. The general fact of effusion of blood in connection with these ulcers rests on a much wider foundation than the few cases where it has been described with microscopic minuteness. Not only are ecchymoses and pigmentations continually associated with the ulcers, but intestinal hæmorrhages, particularly in the submucous coat, are not infrequently observed without ulceration in connection with advanced renal disease, hypertrophy of the heart, and retinal hæmorrhage. I have annexed a table which displays eight cases of this nature.

Besides the ulceration which has been described as belonging to the intestine, I have brought together three cases in which the stomach presented a similar change in similar circumstances. In two of these the stomach was ulcerated together with the bowel, and possibly by a similar process. One of the subjects was a child of eight, an age at which ulceration of the stomach is exceedingly rare. In this instance the gastric ulcer was surrounded by traces of

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old hæmorrhage, while similar signs were abundant about the ulcers in the intestine. Putting aside the evidence of hæmorrhage in the stomach in this instance, the gastric ulcers were not noticeably different from those of common experience.

Putting all the observations together, we recognise in connection with chronic albuminuria a peculiar form of ulceration of the bowel which is associated with, and probably produced by, submucous hæmorrhage; while similar hæmorrhage also presents itself in the same circumstances unattended by ulceration. There is reason to believe that the stomach may, however rarely, participate in the same process.

I will take brief note of what may be called the foreign relations of the intestinal lesion and the association of hæmorrhage in the bowel with hæmorrhage elsewhere. In the total of twenty-two cases where the intestine was ulcerated, with or without the stomach, the retinæ were the seat of albuminuric changes, hæmorrhagic or otherwise, in nine. In the same series hypertrophy of the heart was recorded in nineteen, as if the intestinal lesion were but one of the cardio-vascular series.

Looking at the conditions of kidney found together with this ulceration in the twenty-two cases referred to, advanced fibrosis was present in all but two; in fourteen cases the kidneys were described as granular; in four as fibrotic, or in a state of interstitial nephritis; in two as well-marked examples of the large white kidney of nephritis. Assuming, as we may, that the large white kidneys were affected with interstitial as well as tubal inflammation, it is to be concluded that twenty of the twenty-two were in some phase of advanced renal fibrosis. Reverting to the two exceptional cases where renal fibrosis was not recognised: in one (Powell) the kidneys were certainly the seat of old disease, and were probably fibrotic, though not assuredly so. They were irregular in shape, had adherent capsules, and one presented a cicatricial depression. The other (Louisa Booth) was a case of lar-

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daceous disease which has especial interest in relation to the ulceration of the bowel. The lardaceous disease was not the cause of the ulceration, but its result. There had been general dropsy, presumably renal, at the age of twelve. This appears to have been succeeded by the special ulceration, perforation, and a circumscribed abscess within the peritoneal cavity. The consequent profuse discharge by the bowel led to general lardaceous disease which superseded or masked the renal mischief, and proved fatal at the age of twenty-one.

The symptoms of this complication of renal disease present themselves usually towards the fatal close, though there is evidence in old cicatrices found after death that the ulceration is not always immediately fatal. The symptoms are those of bowel irritation, sometimes such as might belong to dysentery, with which are often associated those of peritonitis and perforation. In the course of these there may be certain reminiscences of uræmia; it is difficult to say how far the occasional obstinate vomiting may have this origin, though other manifestations of the uræmic state appear to be much in abeyance when those of the intestinal lesion are prominent.

The post-mortem observations before us show that peritonitis was present in about a third of the cases, in seven of twenty-two; and that perforation had occurred about half as often, in four of the number. The symptoms of irritation of the bowel are diarrhœa and griping. The diarrhœa is by no means always present; there may indeed be constipation and a need for purgatives. The diarrhœa is sometimes profuse, and the chief cause of the prostration which in these cases is apt to be the mode of death. The motions are often liquid, and like those of typhoid; rarely they contain mucus. The griping pain is often prominent, and is most marked after food. A boy (Charles Dodd), who suffered much in this way and died with peritonitis and perforation, furnished his own diagnosis as "a twisting of the guts." He had served a butcher and acquired the rudiments of anatomy.

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One of the most noticeable symptoms is vomiting, which is often frequent and either spontaneous or readily provoked. A woman (Maria Jackson), under Dr. Bence Jones, had so much vomiting that for a time her disorder was attributed not to the kidneys but the stomach; sickness had been habitually induced by the sight of food and even by the sound of the dinner-bell. In some of the cases recorded it was a matter of doubt how far the vomiting was dependent on the state of the bowels and peritoneum, and how far uræmic. This symptom appeared most prominently when peritonitis was present. Hiccough was occasionally noticed.

Death is usually brought about by prostration and collapse, due either to the diarrhœa or to the peritonitis with or without perforation. Delirium was present towards the close in three of the twenty-two cases. The advent of the abdominal symptoms is usually succeeded by death in no long time; in five cases where this interval was recorded it varied from two days to over two months. In one which has been already referred to (Louisa Booth) there was evidence of albuminuric ulceration a year before death, which led to perforation, a circumscribed abscess, profuse purulent discharge, and lardaceous disease. In a minority of the cases (nine of twenty-two) no bowel symptoms were noted.

Without occupying the time of the Society with considerations which must be obvious, I will rest content with having invited attention, and that in somewhat more detail than before was possible, to a result of chronic renal disease which has hitherto received little notice, but which apparently belongs to a well-known class, for it is presumably of hæmorrhagic origin, is akin to the retinal alterations, and has relation to the cardio-vascular changes of which hypertrophy of the heart is the most constant indication.

The enteric manifestation has interest clinically as well as pathologically, for it commonly foretells the

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approach of the fatal ending, and often brings it about in modes which have been sufficiently denoted.

In conclusion, I have to record my obligation to Mr. William J. Fenton for making the microscopic observations to which I have already referred in connection with his name, and to Dr. Rolleston for assisting him.

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TABLES OF CASES.

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Cases of Albuminuric

Name, physician, date.	Age.	Cause of renal disease.	Duration of renal symptoms.	Urine.	Kidneys, P.M.	Heart, P.M.	Retinæ.
Maria Jackson. Dr. Bence Jones, 1853. Croonian Lectures, 1876	30	—	6 months	Highly albuminous, sometimes bloody; low sp. gr.; casts	Granular, contracted; arteries thickened	L.V. hypertrophied	Much dimness of sight
Charles Dodd. Dr. Dickinson, 1872. Croonian Lectures, 1876	14	Stone? lithotrity at age of 3	3 years	Trace of albumen; casts; pale; low sp. gr.	Granular, contracted, fibrotic; right atrophied from stone	L. V. much hypertrophied; weight 8 oz.	Advanced albuminuric retinitis
Thomas Wilding. Dr. Whipham, 1878	39	A great drunkard; scarlatina in infancy	Œdema 2½ months	Highly albuminous, sometimes bloody	Granular, contracted; weight 8½ oz.	Hypertrophied; weight 18 oz.; recent pericarditis	—
Francis J. Williams. Dr. Dickinson, 'Path. Trans.,' vol. xxix, p. 117, 1878	20	Scarlatinal dropsy at age of 6	14 years (?); polyuria 2 years	Highly albuminous; casts; pale, copious	Granular, contracted, fibrotic; weight 8 oz.	L.V. hypertrophied; weight 15 oz.	Advanced albuminuric retinitis
William Shrapnell. Dr. Whipham, 1878	24	—	2½ months	Highly albuminous; pale	Very granular; weight 11 oz.	Enormous hypertrophy of L.V.; weight 39 oz.; recent pericarditis; pulmonary apoplexy	—

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Ulceration of the Bowels.

Renal symptoms.	Bowel symptoms.	Ulceration, P.M.	Disease bearing on ulceration.
No dropsy; obstinate vomiting throughout, thought to be uræmic; epistaxis	Obstinate vomiting; latterly griping abdominal pain and diarrhœa. Died of peritonitis	Turbid fluid in peritoneum; intestines glued together by recent lymph. Lower half of ileum thickened; showed red and dark patches of discoloration, and patches of ulceration of no great depth covered with thin membrane. Colon natural	None.
No dropsy; frequent headaches; obstinate vomiting regarded as uræmic; convulsions	Obstinate vomiting; griping pains in belly; abdominal tenderness; diarrhœa	Recent peritonitis. Lower two feet of ileum contained many ulcers, some of which were linear and corresponded with valvulæ conniventes; these often exposed peritoneum. Mucous membrane between swollen	None.
Œdema, ascites, vomiting	Latterly diarrhœa and vomiting, with epigastric pains, especially after meals	Small ecchymoses, especially on valvulæ conniventes. In colon, for two feet below valve, small star-shaped and elongated ulcers, the latter transverse; edges congested	None.
No dropsy after original attack; great arterial tension, epistaxis; dyspepsia, vomiting	Nausea, vomiting, griping abdominal pain, tenderness; pain, especially after food; constipation, needed purgatives; mucus in motions	Purulent fluid in peritoneum. Many small ulcers in ileum, mostly in Peyer's patches, clean cut as if punched out, three of which had perforated. Lower half of ileum congested and pigmented. Colon pigmented and ecchymosed. Microscopic sections of ileum showed dark pigment mixed with blood crystals in submucous coat	None.
No dropsy; hæmoptysis, dyspnœa	No local symptoms; constipation, needed purgatives	Numerous ulcers in lower part of ileum and upper part of colon. In colon ulcers numerous near valve. In the middle of the ileum two old, practically healed ulcers, large as Peyer's patches. Recent ulcers small, roughly circular, and with raised edges. Hæmorrhages and pigmentation about ulcers. No peritonitis	None.

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Name, physician, date.	Age.	Cause of renal disease.	Duration of renal symptoms.	Urine.	Kidneys, P.M.	Heart, P.M.	Retinæ.
Frederick Hissgen. Dr. Dickinson, 1879	27	—	2 years	Highly albuminous; sp. gr. 1008	Granular, contracted; cortices shrunk; hæmorrhages in Malpighian capsules	L.V. hypertrophied; weight 16 oz.	Albuminuric retinitis
Philip Parker. Mr. Holmes, 1879	58	A painter	—	—	Very granular; weight 10 oz.	L.V. hypertrophied; R. V. thin; weight 10 oz.	—
Edward Powell. Dr. Dickinson, 1878	21	—	Abscess of hip at age of 8 years; diarrhœa 2 months	Trace of albumen; scanty, high coloured	Both congested; capsules adherent; left irregular in shape, with depressed fibrous patch	Healthy; weight 10 oz.	—
Benjamin Cotton. Dr. Dickinson, 1881	46	Had dysentery in India	Œdema 4 months	Albumen = ½; scanty, latterly bloody	Large, white; weight 21 oz.	L.V. hypertrophied; weight 22 oz.	—

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Renal symptoms.	Bowel symptoms.	Ulceration, P.M.	Disease bearing on ulceration.
No œdema; headache, vomiting	Much pain in belly for 40 days before death, especially about umbilicus, where was tenderness; constipation; hiccough; no diarrhœa; latterly delirium	Peritonitis and extravasation of fæces. Several perforations in ileum and many ulcers in various stages. Ulceration extended from middle of jejunum to middle of ileum. Ulcers generally clean cut; mucous membrane in their neighbourhood covered with thin filmy layer. Tips of valvulæ conniventes apparently first ulcerated. Peyer's patches not especially affected. Colon exempt.	None.
Examination by Mr. Fenton, 1893.—The submucosa is œdematous and swollen from the presence of recently extravasated blood. Around the hæmorrhages are signs of inflammation, *i.e.* small-cell infiltration, probably the result of the hæmorrhage. The vessels are engorged with blood, and the walls of the arteries are thickened. There are some pigment masses in the submucosa, probably evidence of former hæmorrhage. The peritoneum has recent lymph on its surface. The mucosa has entirely gone in parts, with, in some places, part of the submucosa; the removal has the appearance of having been effected by a process of digestion rather than by active inflammation			
Admitted with fracture of thigh; no renal symptoms recognised	Bowels confined at first, then profuse diarrhœa with abdominal pain, tenderness, vomiting, and delirium. Sank from abdominal disturbance, which began 5 days before death	Peritonitis. Descending colon and sigmoid flexure thickened and "worm-eaten." Ulceration more or less continuous in these regions, with many recent hæmorrhages. Bowel nearly perforated in several places. *Postscript*, 1893.—After keeping in spirit punched-out appearance well shown. In many places round, sharp-edged hollows like indentations made by raindrops. *Examination by Mr. Fenton*, 1893.—Submucosa œdematous and swollen by recently extravasated blood. Small-cell infiltration around hæmorrhages, as in case of Hissgen, and probably from same cause. The vessels, especially the veins, distended with blood. Pigment masses present. The mucosa has in places been removed as in preceding case	None.
None beside diarrhœa	Obstinate diarrhœa for over 2 months, with exhaustion, delirium, and death	Several small ulcers of mucous membrane, surrounded by slate-coloured zones of old hæmorrhage, on valve and just above it. Congestion of small intestine and stomach	None.
Œdema; some ascites	No bowel symptoms	Ulcer of the size of a sixpence in small intestine 2 feet above valve; ulcer clean, regular, pigmented. Congestion 6 inches above, but not about, ulcer	None.

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Name, physician, date.	Age.	Cause of renal disease.	Duration of renal symptoms.	Urine.	Kidneys, P.M.	Heart, P.M.	Retinæ.
Sarah Greenfield. Dr. Dickinson, 1882	39	Repeated pregnancies (?)	Headache 6 months	Highly albuminous; sp. gr. 1012; polyuria	Granular, contracted; weight 8 oz.	L.V. hypertrophied; weight 16 oz.	Albuminuric retinitis
Louisa Booth. Dr. Dickinson, 1883	21	General dropsy at age of 12, which lasted 12 months; œdema recurred 4 months before death	11 years (?); 4 months (?)	Latterly albumen = $\frac{1}{3}$; polyuria	Lardaceous; weight 9 oz.	Weight 6 oz.	—
Ann Harffery. Dr. Wadham, 1885	19	Scarlet fever in 1871; nephritis in 1873	8 years	Albumen = $\frac{1}{3}$; casts	Left kidney contracted and fibrotic; right tubal nephritis	L.V. much hypertrophied; weight 10 oz.	—
Ann Mackay. Dr. Wadham, 1885	15	Scarlatina in 1873 (?)	—	Albumen = $\frac{2}{3}$; smoky, scanty; casts	Fibrotic, contracted, pale; capsule adherent; weight 4 oz.	L.V. hypertrophied; weight 8 oz.	—
Ann Shaw. Dr. Champneys, 1888	49	Stricture of urethra (?)	—	Contained blood and pus; alkaline	Both granular; right atrophied; left enlarged and contained abscesses	Old mitral disease; weight 16 oz.	Hæmorrhages in both
George Windover. Dr. Dickinson, 1889	58	Painter; gout	—	Albumen = $\frac{1}{3}$; pale; sp. gr. 1012	Granular, contracted, cysted; weight 6 oz.	L.V. greatly hypertrophied; weight 22 oz.; recent pericarditis	Albuminuric changes in both

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Renal symptoms.	Bowel symptoms.	Ulceration, P.M.	Disease bearing on ulceration.
No dropsy; headache, vomiting	Acute abdominal pain with looseness of bowels 2 days before death	Many pink injected patches on peritoneum. Many prominent congested villi on small bowel, some of which are beginning to ulcerate	None.
General dropsy at age of 12. One year before death showed symptoms of circumscribed abscesses in peritoneum, due (as was subsequently ascertained) to perforation of intestinal ulcers, no doubt due to former albuminuria; then constant discharge of pus from bowels, and consequent establishment of lardaceous disease, which affected liver, spleen, and kidneys. Recurrence of albuminuria and dropsy, with diarrhœa, vomiting, and prostration		Irregular ulcers in large intestine, some of which, in the sigmoid flexure, had perforated the bowel and were in connection with the abscess cavity outside; ulcers irregular in shape, with thin edges. General lardaceous disease, which was apparent during life	None.
No œdema latterly. Died with urœmia and convulsions	Pain in belly 10 days before death, which recurred in severe paroxysms; formerly constipation; diarrhœa latterly, abdomen resonant and tight	Ileum for 6 feet contained ulcerated patches stained with fœcal matter. Ulceration connected with congestion and interstitial hœmorrhage. Some of the vessels plugged by coagula	None.
No œdema at any time; epileptiform fits; urinous smell in breath. Died with convulsions	None noted	Ulcers "near cæcum"—the largest 1 inch by ½ inch. Cæcum and colon present many black patches of the size of pins' heads, due to intense congestion of solitary glands. Congestion present in other parts of bowel, but not to same extent	None.
Œdema, vomiting; Died of urœmia	Vomiting (urœmic?). No distinct bowel symptoms	Large ulcer in middle of transverse colon.	None.
Œdema, ascites	None recognised	Ulcers in transverse colon	None.

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Name, physician, date.	Age.	Cause of renal disease.	Duration of renal symptoms.	Urine.	Kidneys, P.M.	Heart, P.M.	Retinæ.
Henry Webb. Dr. Ewart, 1890	52	Painter	—	Loaded with albumen	Large, white; not lardaceous; weight 16 oz.	Weight 14 oz.	—
Thomas Spaul. Dr. Cavafy, 1890	24	Calculi (?)	3 months	Cloud of albumen; pale; sp. gr. 1010	Both granular; stones in left; left weighed 1 oz.; right 7 oz.	Both ventricles hypertrophied; weight 18 oz.	Albuminuric retinitis; hæmorrhage in right
Henry Slemon. Dr. Ewart, 1890	56	Whiskey drinking (?)	—	Albuminous; scanty, bloody	Granular, contracted; weight 4 oz.	L.V. hypertrophied; weight 12 oz.	—
Jane Moss. Dr. Cavafy, 1890	48	—	3 weeks	Albuminous and lithatic	Fibrotic; contained old infarcts; weight 8 oz.	Both ventricles hypertrophied; weight 16 oz.	Normal
William Jones. Dr. Cavafy, 1891	66	—	5 years	Slightly albuminous; copious, pale	Granular, red, contained caseous masses; weight 12½ oz.	L.V. hypertrophied; weight 29 oz.; pericarditis	—
Ulceration of Stomach possibly Albuminuric,							
Jessie Bevan. Dr. Dickinson, 1888	17	—	2 years	Albuminous; low sp. gr.	Contracted, granular, fibrotic; weight 5 oz.	Great hypertrophy of L.V.; weight 16 oz.	Albuminuric changes

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Renal symptoms.	Bowel symptoms.	Ulceration, P.M.	Disease bearing on ulceration.
Œdema, headache, vomiting, dyspnœa, drowsiness	None noted	Two sharply cut ulcers in first part of duodenum. Mucous membrane of duodenum injected	None.
Headache, vomiting, dyspnœa; œdema latterly	None observed	Ulcer in commencement of duodenum. Cæcum extensively ulcerated, with blood in mucous coat. Old ulcers in sigmoid flexure. Tar-like fæces in colon	None.
Œdema. Died of uræmia. Had also cirrhosis of liver and ascites	None observed	Ulcer in ascending colon. Hæmorrhage in mucous coat of sigmoid flexure	None.
Œdema; delirium	Diarrhœa; motions like those of typhoid	Intestines discoloured externally at intervals. Internally discoloured patches to a marked extent, together with ulceration in the duodenum. A few patches of ulceration in the jejunum. Hæmorrhagic patches in ileum and sigmoid flexure. The hæmorrhages were in the submucous coat, and evidently the primary condition, the ulceration being secondary	Scars at apices of lungs.
Much œdema; mitral systolic murmur; dyspnœa; epistaxis	None observed	One large and two small shallow ulcers in the first part of the duodenum; no thickening; no trace of new growth	None.

with or without Ulceration of the Bowel.

Œdema, headache, vomiting	Epigastric pain after food (ulcers of stomach); much abdominal pain and vomiting	Several small ulcers in *stomach*, the largest about size of threepenny piece. Numerous ulcers from upper part of ileum to lower part of descending colon, in early stage. Mucous membrane thickened and ulcers "punched out." Numerous small brown extravasations beneath peritoneum of ileum	None.

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Name, physician, date.	Age.	Cause of renal disease.	Duration of renal symptoms.	Urine.	Kidneys, P.M.	Heart, P.M.	Retinæ.
Emily Potter. Dr. Dickinson, 1881	8	—	7 months	Albumen = ⅛; alkaline	Interstitial nephritis; unequal in size. Bladder hypertrophied	L.V. greatly hypertrophied; weight 5½ oz.	—
Emma Killick. Dr. Dickinson, 1891	53	—	3 weeks	Albumen = ⅛; lithatic	Red, granular; weight 10 oz.	Hypertrophy and dilation of L.V.; weight 17 oz.	—

Cases of Intestinal Hæmorrhage

Charlotte Rayner. Dr. Cavafy, 1887	40	Pregnancies (?)	7 months	Blood and pus	Granular, contracted; weight 6 oz.	L.V. hypertrophied; weight 17 oz.	Albuminuric retinitis
Edward Heath. Mr. Pick, 1890	42	—	—	—	Highly granular; cortices shrunk; weight 10 oz.	Enormous hypertrophy of L.V.; weight 20 oz.	Hæmorrhages in both
Elizabeth Lucy. Dr. Dickinson, 1890	47	—	12 years (?). 2 months (?)	Solid with albumen; scanty, bloody	Congested; cortices swollen; weight 16 oz.	Weight 12 oz.; recent pericarditis	—
Henry Fowler. Dr. Whipham, 1891	43	Painter; gout	3 months	Albumen = 1/10; pale; sp. gr. 1010	Granular, cysted; cortices shrunk; weight 9 oz.	Hypertrophy of both ventricles; weight 23 oz.	—

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Renal symptoms.	Bowel symptoms.	Ulceration, P.M.	Disease bearing on ulceration.
Swelling of face; no general œdema; wasting; much cardiac tension; gums spongy and bleeding	Vomiting, diarrhœa. Day before death sudden agonising pain in abdomen (perforation)	Recent peritonitis. Local extravasation of fæces. Small intestine from about the middle shows extensive erosions of mucous membrane, with several well-marked ulcers opposite the mesentery; three of these in the last foot of the ileum perforated; no thickening about them. Similar ulcers in cæcum and colon. Intestines deeply stained with extravasated blood; fæcal contents also blood-stained. *Stomach* shows an ulcer near the pylorus, of the size of a sixpence, surrounded by traces of old extravasation of blood	None.
Much œdema; systolic murmur at apex; hæmoptysis; fluid in pleura	"Indigestion" for 3 years before death	Several small ulcers in *stomach*, with several cicatrices of healed ulcers. Surface of stomach congested and covered with mucus	None.

in connection with Albuminuria.

Vomiting	None noted	Small intestine congested, and small patches of hæmorrhage in mucous membrane. No ulcers	None.
Repeated epileptiform fits after a fall	None noted	Numerous hæmorrhagic patches throughout large and small intestines, and also on stomach	None.
Acute œdema; suppression of urine latterly	None observed	Extensive ecchymoses in cæcum. No ulcers	None.
No œdema; asthma; epistaxis. Died of dyspnœa	None observed	Numerous submucous hæmorrhages in ileum. No ulceration	Cretaceous tubercle in right lung.

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Name, physician, date.	Age.	Cause of renal disease.	Duration of renal symptoms.	Urine.	Kidneys, P.M.	Heart, P.M.	Retinæ.
William Werby. Dr. Whipham, 1891	61	—	7 months	Nearly solid with albumen; scanty	Small, white, smooth; not lardaceous; weight 12 oz.	Hypertrophy of L.V.; weight 25 oz.; recent pericarditis	Advanced albuminuric degeneration; hæmorrhages
Richard Seymour. Dr. Dickinson, 1892	46	—	5 months	Albumen = ⅓; scanty	Granular, cysted; cortices shrunk; weight 12 oz.	L.V. hypertrophied; weight 30 oz.; recent pericarditis	Small hæmorrhage in left
John Read. Dr. Whipham, 1892	37	Drink (?)	—	Albumen = ¼; pale	Small, red, granular, cysted; weight 6½ oz.	Great hypertrophy of L.V.; weight 18 oz.	—
John Reardon. Dr. Dickinson, 1892	28	—	1 month	Albumen = ⅓; scanty	Tubal nephritis; congested; weight 13 oz.	Hypertrophy of L.V.; weight 16 oz.	Hæmorrhages and inflammatory effusion

(For report of the discussion on this paper, see 'Proceedings of the

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Renal symptoms.	Bowel symptoms.	Ulceration, P.M.	Disease bearing on ulceration.
Œdema; bronchitis: dyspnœa; uræmic smell	None observed	Small submucous hæmorrhages throughout small intestine. A pigment mass in sigmoid flexure. No ulceration anywhere	Old vomica at apex.
Œdema; arterial tension; dyspnœa	None observed	A few submucous hæmorrhages in ileum. Small pigmented area in ascending colon. No ulcers	None.
No œdema; alcoholic paralysis	None noted	Several ecchymosed areas in ileum. No ulcers	None.
Much dropsy; tense pulse; bronchitis; uræmia	None observed	Lower part of ileum and cæcum show numerous small submucous hæmorrhages. No ulceration or pigmentation	None.

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ABDOMINAL SECTION

FOR

INTESTINAL OBSTRUCTION DUE TO MULTIPLE HYDATID CYSTS.

RECOVERY.

BY

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Received November 14th, 1893—Read January 9th, 1894.

THE subperitoneal areolar tissue is a not infrequent site for the occurrence of hydatid cysts, and it is a well-recognised fact that, in this position, they may exist for very many years without giving rise to any symptoms of note (in one recorded case for thirty-five years). The recto-vesical, or Douglas's pouch, would appear to be the region of the abdomen most commonly involved next to the liver; and even in the former, where the cysts are in immediate proximity to the rectum, they very rarely inflame and suppurate or give rise to trouble, except by their bulk and pressure on the pelvic viscera. M. Tuffier[1]

[1] 'Cinquième Congrès de Chirurgie,' p. 569.

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has collected no less than seventy-four records of cases of hydatid tumours in this region. The interest of the case now reported lies mainly in the fact that intestinal obstruction was produced by three old hydatid cysts and was completely relieved by their removal. There is good reason for thinking that the cysts had existed within the patient's abdomen for no less than twenty-three years; and they well exhibit the remarkable thickness to which the walls of such old cysts may attain. The case is of further interest from the fact that there were only three isolated hydatid tumours in the abdomen, although one was in reality composed of three distinct cysts joined together and in part calcified. Their hardness was greater than that of any tumour except an exostosis, and it may readily be supposed that no correct diagnosis could be made of the cause of obstruction until the abdomen was opened, and that even then at first some doubt was felt as to their nature. The cysts were not directly adherent to the intestine, but were developed in the mesentery.

The patient was a tall thin man aged 53, who had been a gardener all his life. On September 17th, 1893, he was suddenly seized with sickness and abdominal pain; the vomiting continued until his admission to the London Hospital on the 20th. Constipation was present during this time, and the vomiting and pain became more severe. The patient could ascribe the onset to no error of diet or other cause; he had a lobulated tumour in the middle line above the umbilicus some three inches in diameter, and the doctor who attended him suspected this to be a strangulated ventral hernia, and sent him up to the hospital with this diagnosis.

However, on examination, I was sure that the lump was only a protrusion of subperitoneal fat through the linea alba—a "fatty hernia,"—and in no way accounted for his symptoms. There was but little in the previous history to throw light on the case; he had always enjoyed fair health, with the exception of a few "bilious attacks" and the occurrence of ascites at the age of thirty. He was

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then tapped, and after this operation the fluid did not reappear. We may with some confidence ascribe the occurrence of the ascites to the development of the hydatid cysts. The condition of the patient on admission was briefly as follows :—Abdomen somewhat distended, face drawn and anxious, pulse 80, urine acid and containing no albumen. He was repeatedly sick, and the vomited matter was sour and semi-feculent. He was much troubled with hiccough. A free enema only brought away a little clay-coloured fæces. He complained much of pain, especially in the upper part of the abdomen.

Per rectum a hard rounded tumour about the size of a cricket-ball could be felt in the pelvis, and with some difficulty a second one, considerably smaller, could be felt through the abdominal wall below and to the right of the umbilicus.

Abdominal exploration was advised, and being at once agreed to by the patient, was performed on the evening of the third day after the onset of symptoms of intestinal obstruction.

An incision below the umbilicus enabled me to feel distinctly the first of the hydatid tumours, lying amongst distended coils of small intestine. It was of stony hardness, and seemed to be bound down to the peritoneum lining the back of the abdomen ; however, after a time I succeeded in moving it forwards towards the wound, and then found it presented a strikingly white surface, and that it was probably an old hydatid cyst.

The extraordinary hardness made it almost certain that there would be little trouble from hæmorrhage if it were carefully shelled out from the overlying peritoneum, and this proved to be the case, one or two ligatures only being required. After removal of this first tumour a second one was detected just above the brim of the pelvis, and a third, the largest, lay in that cavity. They were dealt with in the same way by peeling off the peritoneum with forceps and the fingers, mainly the latter—necessarily a tedious process, but one attended by no bleeding worth mentioning. Theo-

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retically it would have been well to sew up the rents in the peritoneum, but practically this was impossible, owing to the distended intestine. After satisfying myself that all bleeding had ceased, some warm boracic solution was used to wash the cavity, the parietal wound in the peritoneum was sewn up by a continuous catgut suture, and the muscles and skin by interrupted silkworm-gut sutures. The stomach was washed out, about half a pint of very offensive brown fluid being removed. The symptoms of obstruction were completely relieved by the operation, but for a week the patient was restless and troublesome, being in a curious condition of melancholia alternating with a kind of delirium. His natural temperament was a melancholic one. The wound healed well but for a small abscess in the abdominal wall, due no doubt to inoculation by the patient's fingers, which he would insert under the dressings unless closely watched. There was some diarrhœa for a week or two, but no symptom pointing to peritonitis. He slowly regained strength, and left the hospital at the end of five weeks.

The description of the tumours was as follows:

The smallest of the cysts measured two inches by one and a half, was oval in shape, and, when the patient was under the anæsthetic, could be felt through the anterior abdominal wall; it was placed with its long axis transversely. The second one lay just below it, and was more deeply situated, measuring nearly four inches in length, irregularly lobulated and extremely hard, owing to calcareous degeneration of its wall. The third was pyriform, and lay between the bladder and the rectum (through the latter it had been easily felt); it was freely movable although covered with peritoneum, and shelled out easily.

One striking peculiarity of all of the cysts was the great thickness of their walls—tough and laminated on section, looking like cartilage on their surface; they were about one third of an inch thick. On the inner side was the mother cyst with hundreds of daughter cysts and brood-capsules. The daughter cysts varied in size from

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that of small shot up to that of one inch in diameter, and were closely packed together. In the fluid hooklets of the echinococcus were found, and it was fairly easy to recognise here and there the complete parasite.

The median cyst was composed of three, all with thick walls containing calcareous matter and each containing many daughter cysts.

Little allusion has yet been made to one interesting point—how these hydatid tumours, which had probably existed in the patient's abdomen for some twenty-three years, had suddenly caused intestinal obstruction. The operation did not clearly show exactly where the obstruction was, but it was certainly in the small intestine, and I have little doubt that it was due to the largest of the three hydatids becoming fixed in the pelvis, and thus exercising traction on the mesentery in which they lay. It was almost certainly a mechanical obstruction, there being no trace of inflammation around the tumours. No other hydatids were found within the abdomen, and the case is of interest from this point of view, it being rare for peritoneal hydatid cysts to occur except in a widely disseminated manner. Those who have had much experience of echinococcus disease in Australia tell me that such extremely thick-walled cysts are very rare indeed. Equally uncommon is it for intestinal obstruction to owe its cause to hydatids.

It may once again be pointed out that, with regard to the cysts obstructing the intestine, they formed a sort of chain some eight inches long, reaching from just to the right of the umbilicus down to the bottom of the recto-vesical pouch. They were certainly embedded in the mesentery and not in the great omentum.

In the 'Pathological Transactions' (vol. v, p. 302) is narrated the case of a middle-aged woman who died with symptoms of acute intestinal obstruction lasting for six days. No operation was performed. At the post-mortem an hydatid cyst the size of a large orange was found in the meso-rectum, causing occlusion of the rectum and

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sloughing of its coats by pressure. A number of other cysts were found in the peritoneum of the intestines, uterus, and Fallopian tubes. There were no adhesions. Most of the cysts contained daughter ones. During life one of the cystic tumours could be felt near the umbilicus.

In 1871 Sir Spencer Wells operated on a woman with multiple hydatids scattered about the peritoneum of the intestine and omentum. Only a partial removal was effected, and the patient died within a year, after an illness characterised by vomiting and purging.[1]

I have collected a few other accounts of cases in which hydatid cysts caused death by pressing upon one or other part of the intestinal tract.

In conclusion, I would draw attention to the fact that the presence of a fatty hernia just above the umbilicus led to an erroneous diagnosis of the cause of the intestinal symptoms, probably a not very infrequent source of error. Whether occurring in the linea alba, the femoral canal, or the inguinal region, these protrusions of subperitoneal fat are apt closely to simulate true herniæ. In two papers published in the 'Pathological Society's Transactions' I have collected several instances, and since then have known of other cases in which symptoms of intestinal obstruction occurring in subjects possessing a fatty hernia have been wrongly suspected to be due to the latter. It is a fallacy to be guarded against, especially since it is as a rule both irksome and useless to attempt to retain a fatty hernia by means of truss-pressure. Nevertheless it may be admitted that sometimes the protrusion of the subperitoneal fat draws with it a pouch of peritoneum and so may lead to the production of a true hernia. In the case just described I examined the abdominal wall opposite the supposed hernia from within, and found no peritoneal protrusion.

[1] 'Path. Soc. Trans.,' vol. xvii, p. 278.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. vi, p. 41.)

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DESCRIPTION OF PLATE III.

Abdominal Section for Intestinal Obstruction due to Multiple Hydatid Cysts (J. HUTCHINSON, Jun.).

Case of multiple hydatid cysts causing acute intestinal obstruction. The cysts are represented in the order they were found in the abdomen, the smallest high up, the largest (Fig. 3) in the pelvis.

FIGS. 1 and 2 represent sectional views, and show many daughter cysts within an extremely thick and tough capsule. To the left of Fig. 2 is shown a small secondary cyst which was calcified.

FIG. 3 represents the outer surface of the largest cyst with some peritoneum and vessels attached to it. All the cysts lay in the mesentery.

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Fig. 1.

Fig. 2.

Fig. 3.

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ON THE

(SO-CALLED)

SPONTANEOUS DISAPPEARANCE OF SOLID ABDOMINAL TUMOURS.

WITH THREE CASES.

BY

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Received December 4th, 1893—Read January 23rd, 1894.

My purpose in the following communication is to call attention to a class of solid abdominal tumours which seem to spontaneously disappear. I shall ask your attention only to cases which have come under my personal observation; and I shall offer for criticism a theory as to their origin.

The leading features in the cases to be quoted are, first, the presence of a visible and palpable solid tumour of the abdomen; secondly, total absence of signs of inflammation, local or general; thirdly, diagnosis of malignancy after inspection during exploratory operation; and fourthly, complete disappearance of the tumour with restoration of the patient to health.

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I shall in the first place briefly report the cases.

Case 1.—My first case was met with in June, 1889, and was reported in detail in the 'Lancet' of March 21st, 1891. A summary of the most striking features may here be given.

The patient, a young man of twenty-five, was sent to the Bristol Royal Infirmary on June 1st, 1889, by Dr. Fairbanks, of Wells. He was then suffering from intestinal obstruction. The cause of the obstruction was found on operation to be a solid tumour as large as a cocoanut occupying the left iliac region. An aspirator needle introduced into the growth proved it to be solid. Intestines were in several places adherent to it. It might have been removed, but as the patient was ill from the obstruction, and sarcoma was diagnosed, it was considered advisable to be content with intestinal evacuation and drainage. Enterostomy was performed in the lower ileum, the gut being fixed in the median parietal incision. For about a fortnight nearly all the fæces passed through the artificial opening; but gradually fæces, in increasing amount, appeared by the rectum. When the abdomen was flat the tumour caused quite a marked and localised bulging in the left lower abdomen, and was easily palpable.

At the end of six months it was evident that the intestinal passage had been restored, so the artificial opening was closed by operation. At this time no tumour could be felt through the parietes; and at the time of operation the finger inserted into the abdomen felt only adhesions, but no trace of tumour.

The patient rapidly regained strength and put on flesh; and now after four and a half years remains quite well.

Case 2.—The patient, a widow æt. 50, with grown-up sons and daughters, was under the care of Dr. Davis, of Clevedon. She had been accustomed to lead a very

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active life, and had enjoyed her usual robust health till the spring of 1889, when she observed a gradual failure in her powers. The weakness increased till autumn, when, on October 26th, she fainted on walking uphill on her way from church. Then Dr. Davis was called in. His account of her condition is that he "found her looking worn and ill, with an anxious expression and sallow complexion. There was no rise in temperature. On examining the abdomen, he found a hard dense swelling occupying the umbilical region, adherent to the skin at the umbilicus, and movable with the parietes. At the umbilicus was a small, boggy, uneven swelling, semi-fluctuating and suggesting abscess."

On November 4th, 1889, I saw her, and on November 8th I operated. A small subcutaneous collection of mucopus (about two drachms perhaps) lying under the skin of the umbilicus was evacuated, and the abscess wall was scraped and desepticised. No sinus was discovered. Then the abdomen was opened and the tumour explored. It was perfectly globular in shape, about six inches in diameter, and firm in texture. It rested on the posterior abdominal wall but was not adherent to it, and caused visible bulging of the anterior parietes. Around the umbilicus it was fused with the peritoneum and parietal tissues, and its removal would have necessitated excision of an elliptical piece of the abdominal wall. On the right side and behind, the tumour was smooth and free. On the left side several separate coils of small intestine were closely adherent to the growth, but not embedded in it. To free these intestines would have been almost impossible, and the diagnosis being peritoneal cancer which was becoming diffused, an attempt at removal would have been improper. The operation was therefore abandoned.

A hopeless prognosis was given. The growth was said to be cancerous, and was unremovable because it had invaded the intestines at several points. The diagnosis of cancer was confirmed by the gradual loss of strength and flesh, the sallow complexion, and the total absence of

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pain, elevation of temperature, or other symptoms pointing to inflammatory origin.

However, diagnosis and prognosis were both happily wrong. The patient began at once to improve in general health. A sinus continued to discharge pus for a year; the discharge ceased for six months, reappeared and continued for six months more, and finally healed and has remained closed till now. The patient at the present day is quite well and strong, leading her ordinary active life, and the tumour has completely disappeared.

Case 3 was under the care of my colleague Mr. Harsant, who very kindly gave me abundant opportunities of examining the case, and has given me permission to include it in my report. The patient, a nursemaid æt. 19, was admitted to the Bristol Royal Infirmary on January 9th, 1892. About six months previously she began to complain of feeling weak and ill, and soon after detected a swelling or lump on the right side of the abdomen. The weakness increased and the tumour grew larger, but there was no pain or evidence of disturbance of any vital function. She could eat and sleep well; the bowels acted regularly and naturally; menstruation was normal and there was no loss of flesh. There was only this feeling of weakness, and the abdominal tumour.

The tumour was a solid one, and occupied nearly the whole abdomen, distending chiefly the right side, and apparently in bulk about equal to a uterus pregnant eight months, but rising higher in the abdomen and not dipping into the pelvis. On percussion there was absolute dulness everywhere in front from sternum to pubes, a small area of resonance in the right flank behind, and a large area of resonance in the left flank. On January 11th the growth was exposed by an incision four inches in length. Some odourless cloudy fluid escaped as adhesions between the growth and the parietal peritoneum were separated. No intestine was seen. The growth was closely adherent

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behind and at the top under the ribs, and would have been removed with great difficulty. As, however, it appeared to all of us who saw it to be undoubtedly malignant and to have formed intimate connections with unremovable organs, the operation was abandoned and the wound closed.

On January 19th the wound began to gape, and in a few days a fæcal fistula was formed. Through the fistula all the fæces passed for nearly three months, and simultaneously the tumour gradually diminished in size. On May 1st, the tumour having almost disappeared and there being proof of pervious intestine below the fistula, Mr. Harsant exposed the gut and closed the opening by sutures. On May 6th the wound was found to be quite healed. On June 21st she was discharged practically well, and since then she has continued quite well and strong. There are now no traces of tumour to be felt.

These cases made a strong impression on myself and on all who saw them and watched their progress. Here three solid tumours, the smallest as large as a good-sized cocoanut, the largest not less than an eight months' pregnancy, none of them showing the slightest evidence of inflammatory origin, and none of them being interfered with, disappeared. The diagnosis of malignancy, confidently made on ordinarily accepted clinical grounds, was erroneous. Their evanescence contradicted one essential, almost defining, conception of malignancy. No explanation of their origin and nature that was at all satisfactory could at the time be given.

In my experience the spontaneous disappearance of products that could be at all compared with new growths was limited to myoma, to masses of tubercle, and to inflammatory new growth—peritonitic or cellulitic.

In the case of myoma uteri I have had one where the tumour disappeared after a simple exploration, and two where disappearance followed removal of sloughing portions through the parietes. No doubt similar cases

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have occurred in the practice of most surgeons. It is a well-recognised fact that from trivial causes, physiological or traumatic or therapeutic, fibroids may undergo atrophy or involution. But there could be little doubt that these growths were not composed of inorganic muscle or any tissue allied to muscle, and it would be futile to force the comparison.

So also the analogy of disappearing tubercular peritoneal growths must be dismissed—at least so far as their ordinary behaviour is concerned. For, in the first place, the gross products forming such new growths are removed by physical means, and germicides are introduced. And, in the second place, if the growth re-accumulates and then disappears, such result may usually be attributed to the invasion of a class of micro-organisms which have a liquefying power over inflammatory products and succeed in overcoming the coagulative or constructive power of the tubercle bacillus. A remarkable example of this process is now going on in a patient of mine. After removal of masses of tubercular growth from the abdomen of a child recurrence took place, and a sinus formed through the abdominal incision. Putrefaction occurred during the child's stay outside the Infirmary, and she was readmitted with a discharge from the sinus of enormous quantities of highly offensive pus swarming with saprophytic bacteria. The tubercular growth, in mass at least as large as the child's head, melted away, and now none is to be felt. The reported disappearance or arrest of malignant growth after infection with the products of the micro-organisms of erysipelas has a suggestive bearing on this line of argument. But the reasoning could not applied to these cases without unduly magnifying facts.

Nor, again, had the growths any close affinity with those masses of indurative or "caking" cellulitis which we meet with in the areolar tissues under the peritoneum, and especially in the iliac and pelvic fascia. Such cases of slow indurative cellulitis do indeed sometimes closely mimic malignant growths. Quite recently I was called

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upon to give an opinion as to a case of this sort where a tumour diagnosed after abdominal section by a skilled surgeon as sarcomatous had subsequently greatly diminished in size. In this case an exaggeration of the cellulitic thickening and a minimum of pyrexial signs had contributed to a simple error in diagnosis. Cases of cellulitis, lasting a long time, with little pyrexia, and with sharply defined margins, anywhere in the lower abdomen, and especially in the broad ligament, are liable to be mistaken for tumours, and are sometimes submitted to operation on this diagnosis. A cellulitis in the mesentery might conceivably explain these growths as regards some of their features, but not all.

True peritonitic thickening sometimes reaches enormous dimensions. In the presence of an irritating cause, subacute or chronic, the peritoneum may become thickened to the depth of an inch or even more. In my experience the greatest amount of thickening is found not around a pyogenic focus, but from some source of irritation which is more or less mechanical. And yet the greatest amount of peritonitic thickening which I have ever met with occurred in a case of recurrent appendicitis on which I operated. The patient was under the care of Mr. Forty, of Wootton-under-Edge in Gloucestershire, and the striking feature of it was the presence of a tumour as large as the clenched fist, visible and palpable in the right loin, which could be moved about, and was apparently circumscribed. The amount of peritoneal thickening found on operation was truly extraordinary; in some places it must have been two inches thick. In the centre was found the appendix, the tip of it lying in a little glairy mucoid fluid, perhaps a drachm in all. The appendix was perforated, but it evidently was closed at the intestinal junction, and the fluid which presumably was the cause of the peritonitic thickening was only the proper secretion of the glands in the appendix. It seemed almost a justifiable step in the reasoning to argue from this case to the first of the three which I have recorded, but the step seemed too long to

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pass to the third. And in the case of appendicitis, or protective peritonitis, as it may be called, the circumscription was only apparent, it was not real. The thickening passed between the intestinal coils in all directions, diminishing in bulk as it left the centre, and did not form a simple globe on which the intestines were glued as in the cases under discussion.

Now in such cases as those of plastic appendicitis it ought to be possible to isolate the causes that produce, in some, fine cord-like adhesions; in others, a diffused gluing of the intestinal coils with little thickening; and in a third class, enormous thickening with circumscribed adhesion of coils. When it happened that amongst the cases of plastic appendicitis which I have been called to operate upon, another case with great thickening had also a small collection of glairy muco-pus in its centre, I could not avoid the suggestion of a cause superadded to mere irritation or infection—a specialised effect to meet a specialised cause. It was certainly not a question of simple duration of irritation; the cases with greatest thickening need not be those of longest standing. There was something superadded, and this something seemed to me to be a continuous escape in minute quantities of some extraneous fluid into the peritoneal cavity, and the concomitant isolation and disposal of this fluid by the peritoneal cells.

It seemed that in this way might be explained the origin of these solid tumours. They were simply aggregations of embryonic cells and tissues, heaped up around an intestinal perforation. The perforation was minute, and the amount of escaping fluid was small, so that the cells could effectively deal with it. The perforation remained open for a long period of time, and the continued demand for new cells resulted in an aggregation which was practically a tumour.

In perforation of the appendix the glandular secretion escaped into the general cavity, and the thickening was diffused between the bowels. In these growths the new

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material was definitely circumscribed and globular, and the intestines were only superficially attached to it. To explain this, I inferred that the perforation did not actually go through the peritoneal membrane, but passed through the mucous, fibrous, and muscular coats. And it would be situated somewhere on the gut away from its mesenteric attachment.

The process once started would seem to have an almost unlimited power of extension. The embryonic tissue once vascularised could produce cells at a geometrical rate, and would be capable of dealing with extravasated fluids in greater amount. In the third case the enormous size of the tumour was associated with preparedness to discharge great quantities of fæces through the perforation. In the second case the smaller tumour was associated with a fistula, which discharged only a little mucus. In the third case the necessity for treating an accidental obstruction of bowel concealed any evidence as to the existence of a fistula.

Expressed in terms of modern pathology, the process would be one of phagocytosis. We should say that the power of the peritoneum in its endothelial or its cellular tissue elements to produce microphages and macrophages, leucocytes and plasma cells, was almost without limit, and that the discharge of foreign matter of a certain sort in quantities not too abundant for them to deal with, and continued over a long period of time, might be the cause of this heaping up of phagocytes. The tumour is, in fact, composed of zone upon zone of protective phagocytes heaped up around a minute perforation in the bowel. Whether the source of irritation is purely liquid, or solid particles suspended in liquid; whether it is from an intestinal gland or composed of ordinary intestinal contents, I cannot say, and I should not like to speculate. All that I venture to suggest is that such a cause producing such results as I have described in these three cases would not be out of harmony either with pathological processes or with clinical facts.

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If we accept such a theory, the unscientific postulate of spontaneous disappearance gives way to a simple sequence of cause and effect. In each of these cases the disappearance of the growth was associated with the probable closure of an intestinal fistula. In one there was a sinus discharging muco-pus for two years; the discharge was not fæcal, but it may have been intestinal; it certainly had an external vent. In another, by enterostomy all the intestinal contents were diverted above the tumour, and the supply of foreign matter was cut off. In the third, a fæcal fistula formed, through which the foreign matter escaped, and phagocytosis was not called for.

The mode of cure was in each accidental. It happened that, as nature so often does, in one case the disease had worked its way to the surface, and the surgical division of the skin only accelerated what nature would have succeeded in doing. In another the growth blocked the lumen of the gut, causing obstruction; an operation for the relief of the obstruction happened to cut off the supply, and give the orifice a chance of healing. In the third, some disturbance of the parts rendered easy and safe the completion of the formation of a fæcal fistula already begun, so also giving vent to the foreign matter, and permitting cure to take place.

In conclusion I may be permitted to express my regret that the study of the cases is so incomplete from the pathological point of view. Nothing short of an elaborate post-mortem examination, with careful microscopic study, can provide a full and satisfactory explanation of the origin of such cases. In the meantime I may be justified in bringing them forward, in the absence of any such examination and study, first, because they are evidently extremely rare, and opportunities for observing them post mortem must be still more rare; secondly, because their clinical importance as being so like malignant growths is undeniable; and thirdly, because I am in hopes that the experience of the Fellows

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of this Society will provide more material on which some sound and satisfactory generalised explanation may be built.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. vi, p. 44.)

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ON

NERVE STRETCHING AND SPLITTING

IN

LOCALISED INTERSTITIAL NEURITIS, LEPROUS AND OTHERWISE.

BY

KENNETH McLEOD, M.D., LL.D.

Received November 14th, 1893—Read February 13th, 1894.

In the year 1877, after witnessing in Edinburgh the brilliant results obtained by stretching the great sciatic nerve in cases of sciatica which had resisted all other methods of treatment, and in which grave disturbance of sensibility and serious impairment of movement had existed, I recommended[1] the employment of the same expedient in cases of anæsthetic leprosy. The success achieved in sciatica was attributed by Mr. Chiene to the liberation of the axis cylinders of nerves from the destructive pressure caused by the products of chronic interstitial inflammation. The researches of Dr. Vandyke Carter had shown that the nervous lesion in anæsthetic leprosy is of a similar character; and it appeared to me, presuming

[1] 'Indian Medical Gazette' for July, 1877, p. 190.

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that the explanation of the *modus operandi* of the cure in sciatica was sound, and the pathology of the anæsthesia, atrophy, paralysis, and trophic changes in leprosy correct, that the operation was likely to be followed by relief, if not cure, in such cases. My suggestion was soon put to the test by Dr. Edward Lawrie, who at that time held the office of resident surgeon in the Calcutta Medical College Hospital. Dr. Lawrie reported[1] that he had performed the operation of stretching in thirty cases of ulnar neuritis causing anæsthesia and motor and trophic disturbances. The nature and causation of the lesion was not specified, but it was stated that in every case the operation was followed by benefit likely to be permanent. These cases were treated at the surgical outdoor dispensary of the hospital, and it was remarked that the patients ceased to attend "when the relief they experienced seemed to them decisive." In two cases in which an opportunity was found of watching their after-history, the relief was found to be permanent.

Dr. Edward Downes, a medical missionary practising in Kashmir, where leprosy is very common, put the operation to trial in a considerable number of cases. On the 20th of May, 1886, he read a paper at the Harveian Society of London[2] giving the results of his experience. He had performed the operation of stretching in forty-two instances; thirty-two were well-marked cases of leprosy. He selected the sciatic nerve as the subject of his operations, and the conditions for which he operated were,—anæsthesia of the lower extremities, loss of pigment, tubercles, neuralgia, and ulceration. The duration of the disease in his cases was from one to seven years. "All the cases," he writes, "were benefited by the operation, and in some the ulcers healed completely, and sensation was nearly restored to its normal state. The latter generally took place in the first few days." The success was appreciated by patients,

[1] In the 'Indian Medical Gazette' for September, 1878, p. 229.

[2] *See* 'Lancet,' vol. i of 1886, p. 1070.

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who brought their friends to be operated on. In one case he found that the sensation of the limb continued good for two years; but the great majority of his cases, as is usual in India, were lost sight of. Dr. Downes's successor, Dr. Arthur Neve, writing to the 'Lancet'[1] stated that he occasionally met with cases which had been operated on by Dr. Downes six or seven years previously. Many continued to have good sensation; others returned to have the operation repeated; in a large proportion of cases he found the benefit to be permanent, and in very few indeed was the recurrence rapid or complete. Dr. Neve, on succeeding Dr. Downes in the charge of the Kashmir Medical Mission, continued to practise nerve-stretching in cases of anæsthetic leprosy. In the 'Edinburgh Medical Journal' for November, 1884, he gives a record of 190 operations performed on ninety persons. "In no disease," he remarks, "has nerve-stretching been practised with such uniform success." In eighty-four of the ninety cases improvement and recovery of sensation were noted, in two no improvement occurred, and four died of other causes. He notes that the area of anæsthesia is generally surrounded by a ring or halo of hyperæsthesia, betokening advancing and progressive disease; that the nerves of the trunk are seldom affected; and that the lesions of sensation, motion, and nutrition are generally confined to a definite area corresponding to the distribution of a particular nerve. In seventy-one cases the sciatic nerve was implicated, in seven the ulnar, in five the median, and in two the musculo-spiral. He found that improvement became manifest in from two to five days, that the restoration of sensation took place centrifugally, and that restoration, complete or incomplete, occupied a period of three weeks. At the same time the nutrition of the affected area improved, ulcers healed, and tubercles disappeared. In two cases the opposite limb improved but in a lesser degree. In no case did any detrimental effect ensue, and he observed that the best stretched cases

[1] Of 14th August, 1886, p. 325.

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showed most improvement. Two cases returned after about four months with a recurrence of anæsthesia; in one of these the operation was repeated with benefit. Recurrence was, however, a rare experience; and in many cases which remained under observation for a year or longer, improvement of sensation and nutrition was found to be permanent and progressive. In two cases the ulnar nerve gave way under traction, but in a few days sensation was restored notwithstanding. No local inflammation or suppuration occurred in any case, and normal sensation was recovered in 75 per cent. of the cases.

The operation was sought after by lepers in Kashmir.

I have myself performed the operation of nerve-stretching in many cases of a similar nature, and with corresponding results; and successful cases have also been recorded by Dr. Wallace of Calcutta, Dr. Mitra of Kashmir, Assistant Surgeon Mahendra, Nath. Ohdedar of Srinagar in Garhwal, and others.[1] Indeed, it is evident from dispensary statistics that nerve-stretching has become an established operation in India.

Dr. Beaven Rake of Trinidad has recorded[2] the results of 100 operations of nerve-stretching performed on sixty cases of leprosy. In twenty-six cases the nerve stretched was the sciatic; in eleven, the external popliteal; in forty, the median; in eighteen, the ulnar at the elbow; in four, the same nerve at the wrist; and in one, the supra-orbital. Ulceration was the condition for cure of which stretching was mostly resorted to. As a rule, it is stated, more or less good was obtained. Relief of pain was one of the most satisfactory results. He does not seem to have been as successful in restoring sensibility as Indian operators, but his cases were evidently of a more advanced and aggravated character, in which the trophic disturbance had reached the destructive stage. More or less relief was obtained in forty-seven out of the 100

[1] *See* appended cases.

[2] In the 'British Medical Journal' of 22nd December, 1888, p. 1373.

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operations. The nerve was found to be enlarged in forty-eight instances—principally the median ulnar and external popliteal. In some cases which died of other causes the nerve was examined at the seat of operation. No naked-eye change was seen, nor any adhesions to surrounding parts observed.

Such is a summary of all the evidence which I have been able to collect on the subject of nerve-stretching in leprosy. The operation of *nerve-splitting* was first resorted to by me in the year 1880 in a case which I fully reported in the April number of 'Brain' for that year, a short abstract of which is appended.[1] An East Indian youth, twenty-six years of age, had suffered for eight years from ulnar neuritis, which had resulted in an abscess leaving behind it a sinus occupying the interior of the indurated and thickened nerve. All the characteristic results —sensory, motor, and trophic—of grave disease of the nerve were present in an aggravated degree. I slit up the sinus, which was about four inches long, scraped the interior of it, divided the indurated nerve in two longitudinally, carefully stretched it in addition, and closed the wound, which healed kindly. Improvement in sensation and power commenced in a few days, and continued to progress. I had opportunities of seeing this case from time to time for about ten years, and found on the last occasion that the limb had been restored almost to a normal condition. I have collected eight other cases (six of my own, one by Dr. Bomford, and one by Dr. Tomes)[2] in which the operation of longitudinal splitting was resorted to for localised neuritis with distal impairment of sensation, motion, and nutrition. In two of the cases speedy and signal benefit resulted; in four decided relief was obtained; and in two cases of anæsthetic leprosy in which the neuritis was multiple no improvement took place. I regret that the induction as regards splitting diseased nerves is so limited; but as I shall

[1] No. 1 of Cases of Nerve-splitting.

[2] *See* Appendix.

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have no further opportunity of putting the operation to the test, I have thought it right to represent the matter to the profession as it now stands, in order that further trial may be made by others of nerve splitting in suitable cases.

There are several circumstances connected with the peripheral neuritis of the leprous which appear to me to justify recourse to local measures.

1. The inflammation is, as Dr. Gowers states, "typically interstitial," involving the primary sheath of the nerve and the secondary sheaths of the fasciculi and extending into the interior of the latter among the nerve fibres, which undergo a slow process of degeneration and atrophy from pressure and tension. This process of destruction is progressive, and may continue until the whole of the nerve-fibres composing a fasciculus disappears, and their place is taken by the organised products of the connective tissue inflammation.

2. The neuritis is a focal lesion, being limited to one or a few nerves, and to one or a few points in its or their course. It is a distinctly localised lesion, apt to be multiple; but the less so the more favorable for local treatment. Not only so, but the morbid process seldom, if ever, involves the whole thickness of the nerve. The patchy character of the cutaneous lesion, at any rate in the earlier stages of the malady, indicates this fact, which has also been established by microscopic observation. Dr. Vandyke Carter has clearly shown that the disease may implicate a single fasciculus only or only the sensory or the motor parts of a compound nerve. "During my examination of leprous nerve-strands," he states ('On Leprosy and Elephantiasis,' 1874, p. 52), "no fact impressed me more than that of the limited district of deposit amongst the nerve-tubes; generally those at the circumference, near the perineurium, were most implicated."

3. The lesion occurs only in superficial nerves, and generally at particular places—where they pierce the deep

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fascia, or are close to joints undergoing frequent and extensive movements. The ulnar nerve above the bend of the elbow is one of the most frequently and earliest involved, very often the only one; and it would seem as if the mechanical disturbance due to position, which in health is tolerated, serves, when a proclivity to disease arises, to determine localisation.

4. The inflammatory products undergo similar changes to those which occur in a common inflammation. At first fluid and corpuscular, they in time present the more advanced forms of spindles and fibres; the material consisting in the end of cicatricial tissue, the contractile properties of which are so well known. The affected nerve undergoes changes accordant with these pathological events. At first hyperæmic, swollen, soft, or gelatinous, it eventually becomes firm, hard, anæmiated, or even sclerosed. Increase of size is manifest throughout, but in time the distal part of the nerve undergoes atrophy.

5. Evidence of partial resolution of the morbid process exists. The liquid part of the product may be removed by absorption, and the corpuscular by degeneration; but the latter is prone to undergo progressive organisation. The fact that the bacillus of leprosy is found abundantly in the recent disease, and disappears altogether as the product organises, further indicates that there is a tendency to limitation of the disease in time, or even that a curative or restorative effort arises. Why the resolution is imperfect, and such a strong tendency to organisation exists, is probably due to persisting irritation as in cases of cirrhosis of the viscera. No proof has been as yet adduced of any reproduction of nerve-tubules or endeavours thereto.

6. Evidence of the existence of pressure and tension in the diseased part is furnished by the way in which, on dividing a leprous nerve post mortem, the fasciculi, as shown by Carter, start out of the sheath and stand apart from each other firm and separate; still better by the

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gaping which the wound caused by longitudinal splitting of nerves in the living is seen to undergo.

The degeneration and destruction of the nerve-fibres is evidently a pressure effect, and not, except in the immediate neighbourhood of tubercles and ulcers, a parenchymatous process.

7. This process of degeneration and destruction is a slow, and slowly but surely progressive one. The intensity of the effect-lesion is progressive. There is first, as regards sensation of the distal parts, hyperæsthesia, increased tenderness and tingling pain; then numbness and formication, and finally complete anæsthesia: as regards motion, there is first impairment and stiffness, then abolition, then muscular wasting; and, finally, firm contraction of tendons and immobility of joints: and, as regards nutrition, there is first hyperæmia, effusion, thickening, and change of colour; then glazing, or desquamation, or eruption—papular, eczematous, pustular, or bullous; and, finally, ulceration, atrophy, necrosis, or even gangrene. The area of the lesion gradually extends, and the extension is invariably from periphery to centre. The time comes when, as shown by Dr. Buzzard, the nerve, or a great part of it, loses its nervous elements and becomes converted into a mass of cicatricial tissue. Then it is that the distal portion of it shrinks, and the effects of the destructive process become confirmed and irremediable.

These circumstances give ample and additional countenance to any operative proceeding which may enable the tubules to escape destructive pressure, assist the lesion in undergoing resolution after the crisis of the local disease has passed over, and perhaps permit of salutary restorative as well as recuperative events to occur.

The cardinal objection to operative interference in leprosy is that the disease is of a constitutional character, and that local measures are but palliative and do not in any appreciable manner, or to any material extent, stay the deathward progress of the malady. The truth of this contention cannot be denied; but the symptoms and

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effects of these nerve-lesions are a source of pain and distress, and the evidence which I have adduced makes it abundantly clear that by stretching and splitting nerves affected with leprous inflammation, pain may be relieved, feeling restored, muscular power improved, eruptions cured, ulcers healed, and the process of destruction of the distal parts of the limbs mitigated and delayed. If these benefits can be obtained by operative proceedings which are perfectly simple and harmless, an extremely strong case for their adoption has been established, and the only question that remains is,—under what circumstances, as regards the duration of the disease and of the lesion, and the ascertained or presumed condition of the nerve, the operation of stretching or splitting, or of stretching and splitting combined, may with propriety and promise of success be resorted to. But there are numerous cases, met with more frequently in the tropics, but by no means unknown in Europe and America, as testified by medical literature, in which the surface nerves of the body undergo local inflammatory changes resembling those of leprosy, and entailing consequences as regards sensation, motion, and nutrition, similar to those seen in anæsthetic leprosy. The nerve-lesion may be of traumatic, constitutional, toxic, or miasmatic origin; but whatever the causation, the distal effects are the same. The ulnar nerve is very specially liable from injury or participation in surrounding or neighbouring disease to undergo a process of thickening and induration above the internal condyle of the humerus, and when this condition does not disappear by resolution, the characteristic effects on the sensation, movements, shape, attitude, and nutrition of the forearm and hand are certain to arise, in consequence of pressure effects on the nerve tubules. Cases of non-leprous ulnar neuritis are by no means uncommon, and many of the most brilliant results of stretching and splitting which have been placed on record have been obtained in cases of this kind. In these the arguments which discourage operation in the leprous have no force; and when mea-

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sures such as rest, repeated blistering, galvanism, administration of arsenic and the iodides, &c., have been resorted to without success, and the distal effects are becoming progressively more grave and extending, the question of operation does not, I would urge, admit of doubt or delay.

Whether that operation shall be stretching or splitting or both combined, will, I venture to say, depend on the state of the nerve. If the nerve is found on exposure to be pearly, hard, anæmic, and resistent, I strongly advise a longitudinal incision throughout the whole extent of the diseased part to a depth of one third or one half of its thickness according to circumstances, and a subsequent careful and prolonged but gentle manipulation and stretching of the trunk. There are conditions of nerves in which an accumulation of the liquid products of inflammation takes place beneath the perineurium. A very striking instance of this kind involving the sciatic nerve has been placed on record by Sir Joseph Fayrer in 1868, in which immediate and permanent relief was obtained by incision, and evacuation of about half an ounce of fluid ('Medical Times and Gazette,' vol. i for 1868, p. 8), and he informs me that he has since met with other cases of the same sort. The motive to operate in such cases is different from that which I have endeavoured to impress in this paper; but the relief of pain, numbness, and crippling which was so promptly afforded by incision was undoubtedly due to the removal of pressure and tension; and the signal benefit often conferred by the use of acupuncture in sciatica is doubtless due to the same cause.

From the facts and considerations which I have advanced the following conclusions are, I think, fairly deducible.

1. The operation of nerve-stretching has been followed by decided relief in a large proportion of cases of leprous neuritis which have been subjected to this method of treatment.

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2. The benefits conferred have consisted in the relief of suffering—pain, tingling, &c.—the restoration of sensibility, the return of muscular power, the healing of ulcers, and general improvement in the nutrition of the affected parts. These benefits have speedily followed on the operation, and in a considerable proportion of cases have been sustained and progressive. In many instances repetition of the operation has been attended with success.

3. Additional knowledge is desirable regarding the precise conditions under which recourse to the operation is likely to be advantageous, more especially with reference to the limitation of the local lesion and the stage of the malady.

4. The relief obtained by this proceeding appears to depend on the removal of pressure and tension exercised on nerve-fibres by the products of the inflammatory disease which has taken place in the ensheathing connective tissue of the trunk.

5. When these products have undergone organisation, and the enveloping material has become sclerosed, longitudinal incision of the sheath, followed by manipulation and stretching, give reasonable promise of more complete relief —resolution or even restoration—than stretching alone.

6. This process of splitting sclerosed nerve trunks has been attended with success in a sufficient number of cases to render further trial justifiable or desirable.

7. In cases of non-leprous localised neuritis, these procedures are more likely to be followed by radical and permanent benefit than in cases of anæsthetic leprosy.

8. In no instance has either the stretching or splitting of nerves been productive of any inconvenience or harm.

APPENDIX.

Cases of Nerve Stretching.

Case 1. *Ulnar neuritis; thickening, anæsthesia, paresis; stretching; recovery.*—Hindu male, æt. 40, coolie. Anæs-

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thesia with tingling and pain, of one year's duration, over ulnar area of right hand, the power of which was greatly diminished; skin thickened; ulnar nerve thickened above inner condyle. Nerve stretched. Thirty-six days afterwards, skin healthy, sensation perfect, thickening of nerve gone; tingling and pain disappeared; hand stronger. (Lawrie, July, 1878, 'Indian Med. Gaz.,' p. 229.)

Case 2. *Ulnar neuritis; tingling pain, anæsthesia, and paresis; stretching; perfect recovery.*—European female. Pain, tingling, loss of sensation of left ulnar area; four months' duration; skin discoloured "dull red;" muscles wasted; ulnar nerve thickened and stretched. Two days afterwards pain disappeared, and tingling lost in four days; sensation partially restored. Six months afterwards nerve normal in size, and "functions perfect." No trace of disease existed in any part of the hand, forearm, or arm. Seen by Wallace four years afterwards, "success complete." (Lawrie, October, 1878, 'Indian Med. Gaz.,' p. 270.)

Case 3. *Ulnar neuritis; anæsthesia; paresis; stretching; recovery.*—Hindu male, æt. 45. Had an attack of malarial fever six months ago, during which after a period of tingling the left hand and forearm became anæsthetic except the thumb, forefinger, and half of middle finger. Pustular eruption and blebs appeared over anæsthetic area, and muscles became thin and wasted. Anæsthetic patches existed on dorsum of right hand and left cheek. No history of syphilis or injury. Ulnar nerve above elbow thickened and tender; fingers flexed. Nerve exposed above elbow and thoroughly stretched. In two days sensation began to return. A small abscess formed in the neighbourhood of the wound, and the limb became œdematous; the abscess was opened and the œdema subsided. At the time of his discharge, twenty-three days after operation, sensibility had been restored everywhere except in the little finger and thumb, where it

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remained dull. Muscular power was much improved, and the flexion of the fingers disappeared. He was not seen again after he left the hospital. (McLeod, 1879, 'Indian Med. Gaz.,' p. 229.)

Case 4. *Ulnar neuritis; anæsthesia, &c.; stretching; recovery.*—Hindu male, æt. 25. Anæsthesia of left ulnar area in patches; stiffness of fingers and periosteal thickening of phalanges and metacarpals. Nerve thickened above elbow. Symptoms commenced a year ago with tingling and pain in nerve coincident with ague. A papular eruption occurred, and the skin was thickened and discoloured. Nerve stretched; it had an indurated pearly appearance. Sensation began to return on second day. Improvement progressive; thirty-six days afterwards sensory, motor, and nutritional function of nerve completely restored; twenty-four days later improvement sustained, likely to be permanent. (Wallace, 'Indian Med. Gaz.,' November, 1880, p. 300.)

Case 5. *Ulnar neuritis; anæsthesia; &c.; stretching; recovery.*—Hindu male, æt. 35, coolie. Numbness, preceded by pain and tingling of left forearm and hand; wasting and paralysis of muscles; loss of pigment in patches; symptoms of one year's duration. History of ague; none of syphilis or rheumatism. Ulnar nerve stretched above elbow. Nerve decreased in size; operation followed by pain. Sensation began to return in three days, completely restored in five days, power returning. Improvement progressive. In six weeks sensation and motion had been perfectly restored. (Wallace, 'Indian Med. Gaz.,' November, 1880, p. 300.)

Case 6. *Anæsthetic leprosy; both ulnar and sciatic nerves stretched; marked improvement.*—Hindu male, æt. 30. Anæsthetic patches on trunk and limbs, specially marked in ulnar areas and on legs and dorsal surfaces of feet. Hands wasted; muscular power much impaired.

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Both ulnar and both sciatic nerves stretched; all thickened; right ulnar softened; left sciatic adherent to fascia. Improved rapidly after operation; regained power of hands; sensibility restored everywhere, though not fully. Remained in hospital thirteen days. (Lawrie, 'Lancet,' March 12th, 1881, p. 413.)

CASE 7. *Anæsthetic leprosy; ulceration; right sciatic nerve stretched; ulcers healed and sensation partially restored.*—Hindu female, æt. 40. Anæsthetic leprosy of six or seven years' duration; right leg and foot affected; gangrenous ulcers on toes. Sciatic nerve stretched. Wound healed in a week. Ulcers healed, and sensation was restored to a slight extent. (McLeod, 'Indian Med. Gaz.,' June, 1881, p. 158.)

CASE 8. *Ulnar neuritis; anæsthesia, &c.; stretching; recovery.*—Hindu male, æt. 40. Anæsthesia of ulnar area of both hands, papular eruption on both sides; loss of sensation in left leg and foot. Right ulnar nerve stretched above elbow; nerve much thickened, nearly four times its natural size. Sensation began to return in two days, and was completely restored in five days. Papular eruption disappeared. (M. U. Ohdedar, 'Indian Med. Gaz.,' August, 1882, p. 213.)

CASE 9. *Anæsthesia and ulcer of left foot; thickening of popliteal and musculo-cutaneous nerves; stretching; recovery.*—Hindu male, æt. 40. Toes of left foot became anæsthetic a year ago, and ten months ago sustained wound of sole of same foot which has remained open as a sloughy ulcer. External popliteal and musculo-cutaneous nerves thickened. Anæsthesia involves dorsum of foot, anterior third of sole, and anterior aspect of leg; skin thickened and dusky. These two nerves were exposed and stretched. Sensation was restored, the ulcer rapidly cicatrised and the skin regained its normal aspect and condition. (McLeod, 'Indian Med. Gaz.,' October, 1891, p. 307.)

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Cases of Nerve Splitting.

Case 1. *Ulnar neuritis; splitting and stretching of nerve; marked and sustained improvement.*—East Indian male, æt. 26. Numbness and wasting of left forearm and hand of eight years' duration; sinus above left elbow, result of abscess four years previously. Muscles supplied by ulnar nerve much wasted; griffin hand; sensation diminished or abolished over area of distribution of ulnar nerve up to wrist. Skin congested and thickened; ulnar nerve thickened and hard. Sinus laid open, found to occupy interior of nerve and contain curdy matter, about four inches in length; material scraped out of cavity and out of smaller cavities in the nerve communicating with it. Nerve split in two and thoroughly stretched. Wound healed soundly. Twenty-four days afterwards muscular power and sensation much improved. Fourteen months after this, sensation entirely restored; muscles had regained bulk and power; hand stronger; fingers less bent; ulnar nerves thinner and softer. Seen again ten years afterwards; general health good; sensation undiminished and muscular power much improved; hand almost normal. (McLeod, 'Brain,' April, 1880.)

Case 2. *Double ulnar neuritis; anæsthesia; wasting and paresis; stretching and splitting of right ulnar, and rupture and suture of left ulnar; recovery.*—Hindu, male, æt. 40. Complete anæsthesia of ulnar areas. Muscular wasting and loss of power. Condition worse on left side. Both ulnar nerves thickened above the elbow. Right nerve thoroughly stretched and incised longitudinally. Left nerve nodulated and adherent; broke under traction; ends freed and sutured with catgut. Sensation completely restored on right side, and power returned rapidly. On left sensation began to return two days after operation, and continued to improve until it was re-established in eleven days. Muscular power recovered to a great extent. Returned to work as a road labourer, and

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reported well three and a half months after operation. (Bomford, 'Lancet,' February 25th, 1881, p. 329.)

Case 3. *Anæsthetic leprosy of five years' duration; splitting of left ulnar and stretching of musculo-spiral nerves; no improvement.*—East Indian male, æt. 17. Painful thickening of left ulnar nerve and tuberculated patch of anæsthetic skin on dorsum of wrist and lower part of forearm; commenced with itching of skin five years ago; fingers not affected. Similar patch over right tendo Achillis. General health good. Thickened nerve exposed above inner condyle, split to extent of four inches, and pulled. Sheath found much thickened. Wound of nerve gaped. Operation performed antiseptically on 18th April; healed on 25th November. No benefit. Musculo-spiral exposed above outer condyle and stretched on 28th; wound healed by first intention. Operation followed by wrist drop, which gradually disappeared. No improvement as regards anæsthetic patch; would not have internal cutaneous stretched. (McLeod, 'Indian Med. Gaz.,' June, 1881, p. 158.)

Case 4. *Left ulnar neuritis; hyperæsthesia; splitting and stretching; relief.*—Mahomedan, æt. 52. Two painful patches of thickened skin on wrist and ulnar side of left forearm. Disease of two months' duration. Ulnar nerve split for four inches above condyle and pulled. Sheath pearly in colour and thickened. Hyperæsthesia relieved; thickening of skin reduced somewhat. Wound healed in eleven days; performed antiseptically; catgut drain used. (McLeod, 'Indian Med. Gaz.,' June, 1881, p. 158.)

Case 5. *Left ulnar neuritis; anæsthesia and skin disease; splitting and stretching; improvement.*—Hindu, æt. 29. Ulnar side of left forearm and little finger anæsthetic; skin thickened. Similar patch on left leg above outer malleolus. History of secondary syphilis six

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years ago. Disease of five months' duration; became glazed, red, and discoloured one month ago. Ulnar nerve above elbow much thickened. Nerve split and stretched for about five inches; sheath thick and pearly. Operation performed antiseptically; catgut drain used. Wound healed in twelve days. Thickening of skin gradually disappeared; sensation restored to a slight extent. (McLeod, 'Indian Med. Gaz.,' June, 1881, p. 158.)

Case 6. *Right ulnar neuritis; anæsthesia; history of syphilis and injury. Nerve split and stretched; decided improvement.*—Hindu male, æt. 25. Suffered from syphilis twelve years ago. Got a severe injury of the dorsum of the right hand two years ago, followed by inflammatory swelling and gradual loss of sensation over the inner side of the hand. The ulnar nerve is indurated and thickened above the elbow. The musculo-spiral nerve is also felt to be thickened as it winds round the humerus. There is loss of sensation over the little and ring fingers, as well as over the palm and dorsum of the ulnar side of the hand. Complains of tingling over the inner side of the forearm. There is no eruption or ulcer. There is considerable atrophy of the muscles of the forearm, also of those of the little finger. The little finger is flexed, and the ring finger partially so. The ulnar nerve was exposed to the extent of four inches above the elbow. The coat of it was found to be congested and sclerosed. It was carefully split in the direction of its long axis to the full extent of the wound; it was also stretched. Patient remained in hospital seventeen days. The tingling disappeared; sensation was restored in some measure, and the fingers became more flexible. (McLeod, 'Indian Med. Gaz.,' September, 1887, p. 264.)

Case 7. *Anæsthetic leprosy; multiple neuritis; anæsthetic atrophy; splitting and stretching of ulnar, musculo-spiral, and median nerves; no improvement.*—Jew, æt. 14. Anæsthetic leprosy. Left ulnar, musculo-spiral, and

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median nerves much thickened, and the parts supplied by them quite anæsthetic and wasted. The thickened nerves were exposed, freely split in the direction of their axis, and then stretched. The wounds healed by first intention, but no benefit was conferred by the operation. (McLeod, 'Indian Med. Gaz.,' August, 1888, p. 239.)

Case 8. *Ulnar neuritis, traumatic; anæsthesia, &c.; splitting and stretching; sensation restored.*—Mahomedan male, æt. 45, sustained an injury of right wrist twelve months ago. Six months later the inner side of that hand became the seat of tingling, and that was succeeded by numbness, wasting, and loss of power of muscles, and disease of the skin. Thickening and induration of the ulnar nerve were discovered. The diseased portion was split longitudinally into two and the nerve stretched. Sensation returned in the anæsthetic parts shortly after the operation. Ultimate result unrecorded. (Tomes, 'Indian Med. Gaz.,' August, 1890, p. 244.)

Case 9. *Neuritis of median nerve; anæsthesia; paralysis, ulceration of finger tips; splitting; recovery.*—In this case the enlargement of the median nerves was above the annular ligament. It was fusiform in shape, and very hard. The benefit conferred by splitting this portion of the nerve was early and undoubted. (McLeod, 'Indian Med. Gaz.,' October, 1891, p. 307.)

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. vi, p. 53.)

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TWO CASES OF

INTUSSUSCEPTION OF THE LARGE INTESTINE,

DUE TO THE

PRESENCE OF A PAPILLOMATOUS GROWTH,

SUCCESSFULLY REDUCED BY INTRODUCTION OF THE HAND INTO THE RECTUM, AFTER REMOVAL OF THE GROWTH.

BY

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Received December 5th, 1893—Read February 13th, 1894.

Case 1.—On October 22nd, 1886, I was asked by Dr. J. Mackern to see Mrs. R—, a healthy old lady 84 years of age, for intussusception; and I now quote the history of the case from Dr. Mackern's report. The patient was first seen on October 20th. She was then suffering from dyspeptic symptoms, accompanied by intermittent abdominal pain of a griping character. The bowels had acted, but not in a satisfactory manner. Carminatives and directions as to diet were given.

October 21st.—Pain still present, and more severe. Bowels had not acted. A feeling of nausea existed. A teaspoonful of castor oil was ordered.

22nd.—Sickness followed all food. Bowels still obsti-

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nate. A careful examination of the abdomen was made, when the left side of the abdomen was found to be full, and through the thin abdominal parietes coils of intestine were seen working and twisting about. There was no hernia.

A rectal examination at once showed that there was an invagination into the lower bowel of some part of the tube higher up. Mr. Bryant was at once called in, and his examination brought to light the fact that there was an intussusception into the rectum, and that this was due to the presence of a large papillomatous growth attached to the wall of the orifice of the inner tube—the intussusceptum.

An operation was decided upon on the morning of the following day.

23rd.—Ether was given. The anus was forcibly dilated. The growth and bowel to which it was attached were pulled down below the sphincter and held. The growth was then ligatured at its base in two parts and cut off, and the bowel returned. This was effected by the introduction of the right hand through the dilated anus into the rectum, well above the wrist, when the bowel suddenly rushed away from the hand.

The next morning the patient was comfortable, having lost all her pain and sickness. On the fourth day after the operation an enema was given without any good result. On the fifth day the bowels acted, and from this time the patient made steady progress to recovery, and she never had any trouble with the bowels afterwards. She regained more than her previous strength and took an active interest in everything, being both mentally and bodily surprisingly robust for her age. In October, 1887, or one year after the operation, she was taken ill at St. Leonards, and died on October 31st from paralysis of the left side of her body, in her eighty-sixth year.

Case 2.—On August 7th, 1893, I was asked by Dr. Gilbert Richardson to see Miss R—, æt. 50, who was suffering from obstruction of the bowels of twenty days'

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duration, associated with colicky abdominal pain and tenesmus, with the discharge of mucus from the bowel.

From the report of the case kindly supplied to me by Dr. Richardson, it appeared "that the patient had always been a weakly subject, and had suffered for many years from functional liver trouble, and attacks of gastro-enteric catarrh. The early attacks usually lasted about four or five weeks, and were followed by a period of good health of five or six months' duration, after which a renewal of the symptoms took place. As years passed, however, the attacks became more frequent and more severe. During the attacks mucus was often passed with the motions, and at times this was blood-stained." "During June, 1893," wrote Dr. Richardson, "the patient had been coming to me at weekly intervals, suffering from a condition apparently similar to the usual illnesses, except that more mucus was passed from the bowel, and the treatment employed was attended by less benefit than had formerly been the case." On July 24th, the patient being too ill to leave her home, Dr. Richardson was sent for, when he found her in bed with a temperature of 100°, suffering from a good deal of colicky abdominal pain, restlessness, nausea, and constipation of a week's standing, and as the patient was thin, a hard mass supposed to be a collection of fæces in the ascending and transverse colon was made out to be present. Enemata of oil were ordered, with pills of conium and belladonna, and directions given to report progress to Dr. Richardson. Ten days later, as all the symptoms had steadily become worse, and the means employed had failed to give relief, he was again sent for, when, on making a rectal examination, he discovered the presence of an intussusception just within the reach of his finger, but which, after the lapse of two or three days, had descended to the anus.

I saw the patient at this time (August 7th) with Dr. Richardson, and having obtained the history of the case as just recorded, proceeded to examine her. I found, as I

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expected, a pronounced intussusception filling the rectum, and attached to about half of the orifice of the invaginated bowel a large papillomatous growth (specimen shown). The abdomen was very lumpy, but no distinct tumour could be felt.

The next day, August 8th, the patient being anæsthetised, I proceeded to remove the tumour, and with the kind help of Dr. Richardson completed the operation, including the reduction of the intussusception, in a very satisfactory way.

The operation.—I first of all forcibly with my thumbs dilated the anus to its fullest extent, and then with ring forceps drew down externally the invaginated bowel with the growth attached to it. I next isolated the short peduncle of the growth, and with a needle armed with a thick silk ligature perforated its base so as to enable me to keep it *in situ*, and then, having ligatured the base of the growth in three sections, cut it off. The bowel was subsequently carefully examined, so that no other growths should be left, papillomata being often multiple.

The reduction of the intussusception was then proceeded with as follows:—I first well anointed my right hand and forearm with carbolised vaseline, and with the patient on her left side took the exposed end of the intussusceptum from which I had removed the growth between my fingers and thumb, and returned it within the anus, at the same time by a steady and not very forcible screwing movement of my hand followed the bowel upwards until my hand and forearm beyond its middle had disappeared into the rectum. At first I simply mechanically pushed the intussusceptum upwards, but when I had reached the distance described, the bowel suddenly escaped from my fingers and passed out of reach. I concluded by these signs that the intussusception had been reduced, and so withdrew my hand. In this view I was not disappointed, for after the operation everything went on favorably, all pain ceased, the bowels slowly emptied themselves of their impacted contents, and no complaint

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was subsequently made of incontinence of fæces or more than a temporary anal soreness. Indeed, the patient speedily convalesced, and is now in good health.

Remarks.—I believe the two cases which have just been related are worthy of the attention of this Society, not only on account of their comparative rarity, but from the practical lessons which are to be learnt from their consideration.

I would, however, like to remind you that prolapse of the rectum, and invagination with intussusception of the large or small intestine, are but different degrees of the same condition; and that both are brought about by the same causes, namely, local irritation. The surgeon is familiar with prolapse of the rectum in cases of piles and rectal polypi, as well as in those of ulceration of the rectum and of local irritation by worms; and he meets with the same condition in the rarer but not less marked cases of papilloma of the rectum. He recognises also a certain degree of invagination of the bowel in cases of annular stricture of the rectum, cancerous or otherwise. Here the orifice of the strictured bowel feels to the finger introduced into the rectum either like the exaggerated and patulous mouth of an elongated neck of the uterus, or like a more complete example of intussusception. In the former class of cases the bowel prolapses through the anus. In the latter the upper part of the bowel prolapses as an invagination into the lumen of the canal below.

It would be well for the surgeon to recognise with equal confidence the view that local irritation of any kind, either the result of the presence of a new growth—simple or cancerous, of an inverted diverticulum, or of some other local cause of a more temporary character, when applied for a sufficient length of time to any part of the lumen of the intestinal tract, is prone to be followed by prolapse, invagination, or intussusception; and that these conditions are most liable to occur when the local source of irritation is situated either above and within a few inches of the ileo-cæcal valve—where there is a narrowing of the

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bowel followed by an expansion, or within a few inches of the anus, at which a like narrowing of the bowel exists.

In support of this view I would adduce the well-recognised fact that ileo-cæcal intussusceptions are the most common in young life, when temporary local sources of irritation are most common; and that in the middle-aged and old people, when intussusception and severe examples of prolapse of the rectum occur, some papillomatous growth, if looked for, will very frequently be found; and lastly, that in our museums excellent examples of intussusception due to the presence of papillomata, polypi, and cancerous or other growths are to be met with.

I may add that it was from a full recognition of this view that I was led in both the cases I have brought before you to search for the cause of the intussusception and thus to effect a complete cure. For I conclude that everyone present will accept the opinion that it was from the presence of the papilloma in the bowel, and the irritation it produced, that the intussusception was brought about; and that the intussusception was really due to nature's efforts to get rid of the irritating offending growth. To illustrate this matter further, I have brought from the Guy's Hospital Museum some specimens.

The first (1819[95]) is one of intussusception of the *small intestine* about three feet above the cæcum, due to the presence of a polypus the size of a chestnut with a broad pedicle. It was taken from the body of a woman aged forty-two, who died after an illness of ten months under the care of the late Dr. Moxon, and who had suffered from gnawing pains at the umbilicus for nine months previously, associated with diarrhœa, vomiting, and abdominal distension.

The second (1849[18]) shows a *jejunal intussusception* with polypoid growth at the apex of the intussusceptum, taken from a young woman, aged nineteen, a patient of Dr. Goodhart, who suffered from periodical attacks of vomiting and abdominal pain for nearly two years before

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her death, and in whom a kidney-shaped tumour was felt in the lower part of her abdomen, which towards the end of her illness was observed to undergo slow rhythmic alterations, being alternately hard and well defined and soft and ill defined.

The third (1819[45]) is one of an *inverted diverticulum* of the ileum causing intussusception, taken from James C—, aged twenty-two, who was admitted under Dr. Fagge for constipation and vomiting of five days' duration. He was operated upon by laparotomy and the intussusception was reduced, but he died a few hours after the operation.

Three are examples of cancer of the colon associated with intussusception, viz.:

Specimen 1849[17], which was taken from a woman aged fifty, who suffered for months before death from chronic intestinal disturbance and a lump in her right flank, and passed also a large shred of sloughing tissue. The intussusceptum, which was of the colon, was enormously thickened, and at its apex there is a sloughing mass of growth attached by a slender pedicle.

Specimen 1875[5], which is a portion of colon in a condition of invagination. The intussusception is about four inches in length. The wall of the intestine at the returning angle is greatly thickened, and the mucous membrane of the entering layer is partially destroyed by ulceration. Histologically the wall of the gut is infiltrated by a growth of cylindrical-celled carcinoma.

Specimen 1887[75], which is one of adenoid cancer of the rectum causing intussusception, taken from a man aged forty-four.

In both the cases I have recorded, the cause of the intussusception was the presence of a papilloma of large size; in both the papilloma had a broad base, and involved only a segment of the circle of the intestinal lumen; and in both it was quite certain that the disease was situated high up the bowel, that is, at a far distance from the rectum, for in Case 1 when the intussusception was reduced after the removal of its cause I had to introduce

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my hand well beyond the wrist to carry out my object, and the bowel then sprang out of my fingers with a rush, like that of the intestine in a case of reduction of strangulated hernia by the taxis; and in Case 2 I had to insert my hand and forearm nearly up to the elbow before the intussusception of the involved bowel escaped upwards from the hollow of my fingers in which it rested.

It seems also from the two cases, and from the preparations I have brought before you, that a growth which involves only a segment of the circle of the intestinal lumen is more likely to be associated with a complete intussusception than an annular stricture, as it is certain that these papillomata when they attack the rectum are attended with far more straining, tenesmus, the discharge of serous fluid, and prolapse than any other growth, whether cancerous or otherwise.

There is no need to say much as to the treatment of these cases, for it was such as the judgment of any good surgeon would support when the diagnosis of the case had been determined upon; but surely its success is suggestive of a lesson we might well take to heart, and should lead us to apply the means which in these two cases proved so successful to future examples of intussusception in adult females which have made their way into the rectum, whether due to the presence of a growth or to some unknown cause.

In many cases it is more than probable that success would not follow, but in some it surely would be achieved; in the only two examples in which I have employed it, or I think I may say in which it has been employed, the result has been all that could be wished.

In conclusion, I may add that I found little difficulty in introducing my hand into the rectum after I had forcibly dilated the anus. A steady half-screwing movement, alternating with moderate pressure, effected the desired purpose, and enabled me to pass the anal orifice of the bowel as well as the narrowing of the bowel at the brim of the pelvis, and these two points being passed, all difficulty vanished.

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I have never succeeded in passing my hand into the rectum of a male patient, but have never failed in the case of the female adults upon whom I have made the attempt —about a dozen in all—and my hand when closed for introduction measures over the knuckles nine and a quarter inches.

I may add that in neither of these cases, nor in more than one of the others in which I have introduced my hand into the rectum, has any very prolonged want of control of the anal sphincter been complained of.

In thanking the Society for their kind attention to this paper, I would strongly urge upon surgeons the adoption of the practice which it illustrates.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. vi, p. 57.)

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CASES

OF

PLEURISY CAUSED BY THE PNEUMO-COCCUS,

AND WITH

CONSTITUTIONAL SYMPTOMS RESEMBLING THOSE OF PNEUMONIA.

BY

J. W. WASHBOURN, M.D., M.R.C.P.,

ASSISTANT PHYSICIAN TO GUY'S HOSPITAL; PHYSICIAN TO THE LONDON FEVER HOSPITAL.

Received December 27th, 1893—Read February 27th, 1894.

Case 1.—Mr. L. E. C—, æt. 23, medical student. Admitted into Guy's Hospital on January 8th, 1893, under the care of Dr. Washbourn.

Previous illnesses.—Bronchitis at three years of age; herpes zoster at eight; scarlatina at ten.

History of present illness.—On January 6th, while going round the wards of the hospital, he was suddenly seized with a rigor and was compelled to go home. Next day he suffered from pain in the right side attended with fever. He was seen by a medical man, who diagnosed pneumonia, and recommended him to go into the hospital. On the following day, January 8th, he was admitted.

Condition on admission.—He is obviously very ill and in much pain. The pain is aggravated by a troublesome

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cough, and by any effort to move. The respirations are rapid and shallow, fifty per minute. The sputum is scanty, and consists of mucus which is not rusty. The tongue is covered with white fur, and the bowels are confined. The pulse is 133, weak and compressible. The urine is high coloured, sp. gr. 1024, and contains a trace of albumen. An examination of the chest revealed dulness at the right base behind, with crepitations and deficient breath sounds. A pleuritic rub was audible in the right axilla. The temperature was 102·6°.

For the next four days the general condition remained unchanged. The temperature continued high, and on one occasion reached 103·4°. The respirations varied between 40 and 60. The base of the right lung remained dull; loud râles and occasional rhonchi were audible.

On January 13th a slight improvement in the general condition was observed. The pain was less, the temperature was lower, and the respirations had sunk to 30 per minute. The improvement continued; and on January 16th the pain and cough had disappeared, the respirations were only 22 per minute, the pulse was 80, and the temperature nearly normal.

On January 17th the respirations were 20, and the patient was considered to be convalescent. The physical signs in the lung, however, remained unchanged.

On January 18th the temperature, which had never been quite normal, went up to 101°.

During the next five days the temperature oscillated between 101° and 102°, and the respirations between 22 and 28. The physical signs, which had never cleared up, gradually pointed definitely to the presence of fluid in the chest.

On January 24th a needle was put in the chest and pus found. An anæsthetic was given, a rib was resected, and one pint of pus evacuated. From this time the temperature began to fall, and the patient made a rapid recovery, and is now in perfect health.

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Examination of the pus.—This was examined microscopically, by cultivations, and by inoculation experiments, and was found to contain the pneumococcus of Fraenkel.

Remarks.—This case was at first looked upon as one of pneumonia followed by an empyema. The points in favour of pneumonia were the suddenness of onset—beginning with a rigor, the high temperature, cough and rapidity of breathing. The disappearance of the constitutional symptoms on the tenth day, while the physical signs remained unaltered, pointed the same way. The physical signs were, however, not typical, and the sputum was never characteristic.

Looking at it in the light afforded by our next case, the probability is that it was one of primary empyema, and that the lung was not affected at all.

Case 2. (I am indebted to Dr. Hale White for permission to publish this case.)—J. B—, æt. 36, fireman. Admitted into Guy's Hospital on Nov. 21st, 1893, under the care of Dr. Hale White.

Previous illnesses.—Rheumatic fever three times, at the ages of thirteen, fifteen, and seventeen. "Low fever" twice, once at Singapore and once on the East Coast of Africa.

Present illness.—On the morning of November 15th he was feeling in exceptionally good health, but in the afternoon he suffered from a slight cough. On November 16th the cough increased, and was accompanied by headache and rigors. On November 18th he took to his bed, and remained there until November 20th when he felt better. On this afternoon he was summoned to a fire, and not having permission to stay away, he attended. A doctor had sent him medicine under the impression that he was suffering from influenza. On the evening of November 20th, after returning from the fire, he was rather suddenly taken decidedly worse, and from his account probably had a rigor. On the next day he was admitted into Guy's Hospital.

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Condition on admission.—He is prostrated. The breathing is rapid and shallow, 46 per minute. There is a troublesome cough accompanied by an expectoration of tenacious mucus, which, however, is not definitely rusty, although one specimen was doubtfully so. The tongue is furred, the temperature 104°, and the pulse 102. The urine has a specific gravity of 1032, and contains a little albumen. An examination of the chest revealed the presence of a systolic bruit over the cardiac area. Both sides of the chest moved equally. There was a patch of dulness on the right side, in the neighbourhood of the angle of the scapula, over which the breathing was bronchial, and bronchophony was present. Below this the note was not good, but it did not differ much from that on the other side. Tactile vocal fremitus was equal on the two sides. At the lower part of the right side there were râles and a deficient entry of air. A few rhonchi were heard over various parts of the chest.

A diagnosis of pneumonia following upon influenza was made.

November 22nd.—Herpes has appeared on the lips. The temperature remains at 104°. The physical signs are much the same, except that the râles at the angle of the right scapula are consonating.

23rd.—Delirium. Pain in right side of chest, temperature 102·6°, respiration 50, pulse 108.

24th.—Delirium continues. Temperature 103·4°, respiration 40, pulse 120. There is dulness all over the right chest behind, and this is the first day that the right base has been completely dull. Loud bronchial breathing is audible at the angle of the scapula. Venesection was performed and two ounces of blood abstracted.

25th.—Much worse, pulse 150, temperature 103°. Is very delirious, and has to be held down in bed. Examination of the back of the chest was impossible. Gradually got worse, and died at 8 p.m.

Remarks by Dr. Hale White.—This case was looked upon as one of pneumonia on account of the history, which

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looked like that of pneumonia following influenza, and on account of the persistent high temperature, rapid breathing, cough, herpes, delirium, the rigor at the commencement, and the general aspect of the patient, which was commented upon by three physicians as being typical of pneumonia. The physical signs, although not quite typical of pneumonia, were not more atypical than they often are, and unfortunately they did not suggest a large amount of pus. For on admission there were signs of consolidation at the angle of the scapula, below that the note was only impaired, tactile vocal fremitus was present, râles could be heard, and both sides moved equally; and it was not until the day before death that the right base became quite dull. Also the temperature never oscillated unduly, but kept more or less constantly high.

At the post-mortem examination made by Dr. Fawcett, the following conditions were observed :—The heart was enlarged and the mitral valve thickened and the orifice contracted. The kidneys were tough, but otherwise healthy. The right pleura was covered with recent lymph, and the cavity contained 54 oz. of pus. The lower lobe of the right lung was completely airless and tough, but there was no inflammatory consolidation. The upper lobes were œdematous and congested. The left lung was œdematous and tough, the latter condition being probably due to the condition of the heart. Microscopically the lung tissue was found to be normal.

Examination of the pus.—This was found microscopically, by inoculation, and by cultivations to contain the pneumococcus of Fraenkel.

This case, then, was one of acute empyema caused by the pneumococcus, with constitutional symptoms resembling those of croupous pneumonia.

Case 3.—L. M—, æt. 2 years and 8 months, admitted into Guy's Hospital on August 28th, 1893, under the care of Dr. Washbourn.

History of present illness.—Mother noticed that the

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child was languid and unwell on August 22nd. This continued until August 25th, when she was first attacked with cough, and the face was observed to be puffy and the feet swollen. She got worse, and was admitted into the hospital on August 28th.

Condition on admission.—Patient is breathing rapidly and has a cough. Respiration is 52 per minute, the temperature is 103°, and the pulse 140.

There is dulness at the right base with deficient entry of air. Râles are heard over the upper part of the chest behind, and there is bronchial breathing at the upper part of the dull area.

The feet are œdematous and the urine contains albumen with hyaline, epithelial, and blood casts.

On August 29th a needle was inserted into the lower part of the chest, and a little turbid fluid removed. The fluid examined microscopically showed the presence of leucocytes and diplococci. These latter were proved to be pneumococci by inoculation and by cultivations.

On August 31st the dulness increased, and the chest was aspirated and 4 oz. of fluid removed. The fluid was found to be similar to that previously obtained, and pneumococci were found. The removal of the fluid gave slight relief.

On September 1st there was dulness all over the right chest, and a rub was audible on the left side.

On September 2nd the constitutional symptoms increased, and death took place.

At the post-mortem examination, made by Dr. Shaw, the following conditions were observed:—The whole of the right pleura was found to be covered with thick, tenacious fibrin, and the cavity contained 6 oz. of turbid fluid; the lower lobe of the lung was solid from compression, but there was no inflammatory consolidation. There was recent fibrin over the left pleura. The kidneys were but little altered; the cortex was slightly speckled, and the pyramids perhaps a little congested.

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Remarks.—These cases present several points of interest. It is well established that the majority of cases of croupous pneumonia are caused by the pneumococcus, and the same micro-organism has been found in many cases of empyema following pneumonia. The fact that the pneumococcus when invading the pleura produces the same constitutional symptoms as if the lung itself were involved, has however, not been sufficiently emphasised. The author believes that Case 2 clearly shows that this is true. In this case there was herpes, high temperature, delirium, cough, and rapid breathing, and the patient had all the appearance of one suffering from pneumonia. Yet at the post-mortem examination the lung was found to be unaffected. The constitutional symptoms were doubtless caused in the same way as in pneumonia, *i. e.* by the absorption of the toxins produced by the pneumococcus.

It may be incidentally mentioned that the blood-serum obtained from this patient was found to possess properties similiar to those which, the author has reason to suppose, are possessed by the blood-serum of patients suffering from pneumonia.

In Case 1 it is possible that the empyema may have been secondary to pneumonia, but it should be remembered that the physical signs were not conclusive, and the sputum was not characteristic. The fall of temperature and the disappearance of the constitutional symptoms on the tenth day would receive the same explanation, whether the lung or the pleura were primarily attacked.

The secondary rise of temperature on the eleventh and following days can be explained by the absorption of the inflammatory products.

Case 3 is of interest, inasmuch as the constitutional symptoms were similar, but the pleurisy was of a fibrinous character. Such a case might have recovered, and have been looked upon as one of pneumonia.

The presence of albumen in the urine in the first two cases and of blood in the third is of interest.

In pneumonia albuminuria is common, and distinct

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evidence of nephritis not rare. This is also in accord with the experience gained by the author as the result of inoculation with the pneumococcus. The author is thus led to believe that many cases which are diagnosed as pneumonia, but in which the physical signs are not typical, are really cases of invasion of the pleura by the pneumococcus. Recovery by crisis before the physical signs have cleared is in no way opposed to this view.

The fact that the pneumococcus may at one time produce a fibrinous inflammation, and at another a suppuration, is in accordance with experimental evidence.

Lastly, the author would venture to point out the necessity of exploring the chest in cases simulating pneumonia but with equivocal physical signs.

He would also point out the value of a bacteriological examination of the pus in cases of empyema. The prognosis is favourable if the empyema is caused by the pneumococcus, and it is probable that these are the cases which are cured by a simple aspiration.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. vi, p. 61.)

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RUPTURED GASTRIC ULCER

TREATED BY

LAPAROTOMY, GASTRIC SUTURE, AND WASHING OUT OF THE PERITONEUM;

RECOVERY.

BY

THOMAS H. MORSE, F.R.C.S. (Norwich).

(Communicated by Mr. BARWELL.)

Received January 3rd—Read March 13th, 1894.

Miss C—, æt. 20, had for several months past suffered from pain immediately after taking food, so much so that she had learnt to avoid taking certain things, such as meat and strong tea. She had been somewhat anæmic, but with this exception she had no history of previous illness, and there had been no attack of hæmatemesis. There was no family history of phthisis. Her occupation was that of a draper's assistant.

The present illness commenced as follows:

On December 7th, 1893, having just finished tea, consisting of bread and milk, currant pudding, and tea, she was stooping down to lace her boot, when suddenly she was heard to scream, and said she had most violent pain

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in the abdomen; then followed faintness and vomiting of the food just taken, but the pain continued. Some brandy was given, which very much increased the pain.

When I saw her shortly afterwards she was in bed, lying on the right side with the knees drawn up, moaning with pain, very cold and pale; the respiration was shallow and quick, 30 per minute, the pulse feeble, 80 per minute. She said she could not get her breath, as inspiration gave so much pain.

On examination the abdominal walls were found to be very rigid. She located the origin of the pain at a point just to the left of the middle line and beneath the free border of the ribs; pressure here increased it, and as I watched her during the next few minutes she described the pain as unbearable, of a burning nature, and as extending down towards the pelvis and all over the abdomen. On examination *per rectum* all the pelvic organs proved to be normal in size and freely movable. This, together with the fact that the lips and gums remained a good colour, put aside any suggestion of internal hæmorrhage. I came to the conclusion that it was a case of ruptured gastric ulcer, and advised abdominal section without delay.

Dr. Burton-Fanning and Mr. Walters saw her with me shortly afterwards, and agreed in the diagnosis and treatment. A quarter of a grain of morphia was given subcutaneously. Dr. Burton-Fanning gave ether; Mr. Walters assisted me; it was now nearly five hours since the commencement of symptoms. The abdomen was opened just to the left of the middle line above the umbilicus, the upper extremity of the incision being placed at the lower margin of the cartilages of the ribs, and extending downwards for three inches. As soon as the peritoneum was opened, the contents of the stomach were seen to be scattered all over the intestines; they consisted chiefly of milk with some bread crumbs and currants mixed with mucus. The whole stomach was then withdrawn from the opening, and wrapped up in hot sponge-cloths; both its surfaces were searched for a perforation, which after

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some difficulty was found high up under the ribs on the anterior surface, and close to the cardiac orifice; it was about the size of a cedar pencil, and quite circular.

I dilated it sufficiently to admit one of Lawson Tait's half-inch cannulas used for washing out the abdomen; through this I removed all the remaining contents of the stomach by alternately filling it with water and squeezing it out again.

I then, with a series of Lembert's sutures of chromic gut, introduced with a curved needle, completely closed the opening by inverting a piece of the stomach wall so as to bring into contact its peritoneal coat over a space nearly two inches broad, that is, one inch on either side of the perforation; the stomach was then replaced within the abdomen. I next proceded to wash out of the peritoneal cavity the matters which had been extravasated. To facilitate the return of the water, I passed in by the side of the tube of the irrigator a long half-inch glass tube, and by moving these two tubes about together I went systematically all over the abdomen until the water returned from every point quite clear. As much as seventeen pints of hot water (temp. 105°) were used in this way. I then put in a long glass tube downwards towards the pelvis, and united the wound with fishing-gut sutures and dressed it.

The operation lasted an hour and twenty minutes. The patient bore it well, and by the end of it the pulse had improved considerably. After waking up from the anæsthetic she at once went off to sleep, and slept for two hours, after which she expressed herself as comfortable and almost free from pain.

The after-treatment consisted in giving nothing at all by the mouth for the first sixty hours. During this time enemata of peptonised milk, peptonised beef tea, and warm water were given in succession at intervals of six hours, commencing with five ounces, and, as this was well borne, increasing the amount to ten ounces, and adding also one ounce of brandy. Besides this one of Slinger's

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nutrient suppositories was given every twelve hours. All of these were retained.

The drainage-tube was left out after twenty-four hours, and feeding by the mouth was commenced most cautiously after sixty hours as follows: First water only was given, two drachms every hour, which was soon increased to four drachms every hour. At the end of the next twelve hours she was taking one ounce of milk and one of water every two hours. Seventy-two hours after the operation she was taking four ounces every three hours, consisting of milk and water, milk and barley water, and beef tea. None of these gave any pain. By degrees she had more and more, and on the fifth day she had farinaceous food, and fish on the seventh day. The remainder of her convalescence was uninterrupted.

At the end of three weeks the wound had quite healed, and she was allowed to get up for half an hour. Neither this nor the taking of food gave any pain at all, and in fact she was then quite well.

I wish to call attention to the fact that washing out the peritoneal cavity with a large quantity of hot water relieved the condition of shock, partly no doubt by the heat thus conveyed, but more especially by removing the irritating material, viz. the contents of the stomach.

I am not aware that up to the present time there has been a single other successful case of this operation in this country, when performed, as in this case, for acute perforation of the stomach. Unsuccessful cases have been reported in the 'British Medical Journal' for the past year, 1893, where the subject has been fully and ably dealt with by Mr. Barling, June 17th, and by Mr. Haslam, November 11th.

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Cases of Ruptured Gastric Ulcer in which, after laparotomy, the perforation was found and closed by suture.

Name of operator.	Date.	Time that elapsed between perforation and operation.	Publication.	Result.
Taylor, J. W.	1887	24 hours	Birming. Med. Review	Died.
Czerny	1887	2 days	Internat. Med. Mag.	Died.
Kriege	Oct. 24, '88	57 hours	Rev. Internat. Therapeut. et Pharm.	Died in 10 days.
Mickuliez	June 23, '89	24 hours	„ „	Died in 24 hours.
Kriege	May 20, '92	17 hours	„ „	Recovered.
Steinthal	—	50 hours	„ „	Died in 9 hours.
Stelzner	—	18 hours	„ „	Died in 10 days.
„	—	48 hours	„ „	Died in 4 days.
Le Dentu	—	4 days	„ „	Died.
Taylor, J. W.	Nov. 1, '90	24 hours	Prov. Med. Journ.	Died in a few hours.
Weir	Jan. 18, '91	4 days	Internat. Med. Mag.	Died.
Gilford	Oct. 19, '92	10½ hours	Brit. Med. Journ.	Died in 4 weeks.
Anson	Sept. 12, '92	24 hours	Lancet, 1893	Died in 6 hours.
Haslam	Feb. 2, '93	3½ hours	Brit. Med. Journ., 1893	Died.
Barling	—	6 days	Brit. Med. Journ.	Died.
Morse	Dec. 7, '93	5 hours	—	Recovered.

In addition to the above, several cases have been reported in which the edges of the perforation were sutured to the abdominal wound, *vide* Clinical Society's 'Transactions,' vol. xxvi, a case by W. Lee Dickinson and Warrington Haward.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. vi, p. 70.)

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A CASE

OF

RESECTION AND IMMEDIATE SUTURE OF INTESTINE

WHICH HAD BEEN

STRANGULATED EIGHTY-ONE HOURS; RECOVERY.

BY

C. B. LOCKWOOD, F.R.C.S.,

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SURGEON TO THE GREAT NORTHERN HOSPITAL, &C.

Received January 16th—Read March 13th, 1894.

In March, 1891, I had the honour to communicate to the Royal Medical and Chirurgical Society a paper on the treatment of strangulated hernia when the intestine is gangrenous or ulcerated.[1] I now venture to present the result of the first opportunity I have had of practising what I then advocated, namely, resection and immediate suture of the damaged intestine. This can be briefly done because the literature of the subject has been so recently collated by myself and Mr. Franks.

The case itself has a claim to attention because I believe that this operation has not hitherto been successful after such a prolonged period of strangulation as eighty-one hours. Mr. Kendal Franks, in the exhaustive paper which he read last year,[2] and in which he recorded

[1] 'Medico-Chirurgical Transactions,' vol. lxxiv, p. 199.

[2] "On Resection of the Intestine and Immediate Suture in the Treatment of Gangrenous Hernia," 'Medico-Chirurgical Transactions,' vol. lxxvi, p. 222.

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the results of two hundred and twenty-two similar operations, says "the duration of strangulation did not exceed three days in any of the cases which recovered after resection and suture, with one exception, a remarkable case of Littre's hernia, in which the strangulation had lasted for seventeen days." Nor can I ascertain that Zeidler,[1] who has recently collated five hundred and seventy-six cases, mentions a recovery after such protracted strangulation.

The details of my case are briefly as follows:—The patient was a robust youth, æt. 17 years, who was a greengrocer. He had always enjoyed good health, and until the present occurrence had had no symptoms of hernia. On July 24th, 1893, he had his midday meal at twelve o'clock, but afterwards began to vomit and have pains and dragging sensations in the right hypogastric and inguinal regions. During the afternoon he vomited four or five times and consulted a medical man, who gave him some medicine and some lotion to apply to a swelling which there was in the right side of the scrotum. After the sickness had begun he ceased to pass either flatus or fæces *per anum*. On Tuesday, June 25th, and Wednesday, July 26th, there was no amelioration of these symptoms. Taxis was applied unsuccessfully upon the Wednesday, and again on Thursday, July 27th. Upon the evening of this last day I was called to the Great Northern Hospital to see the patient. The symptoms of intestinal obstruction were typical and very acute. There was no passage of flatus or of fæces; there was continuous stercoraceous vomiting; and the abdomen was distended. The right side of the scrotum and the right inguinal canal were occupied by an acutely strangulated, complete inguinal hernia. The left testis was undescended, with an interstitial hernia, which could be easily reduced. The skin over the tumour was hot and dusky, and the hernial sac was very tense. The general condition of the

[1] "Ueber Darmresektion bei gangrän in eingeklemmten Brüchen.," 'Cent. für Chirurgie,' January 21st, 1893, p. 62.

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patient was serious. He was restless; pale and anxious; his cheeks were hollow and his eyes sunken, with black rims around them; and his pulse rapid and thready. It was inferred that the case was one in which intestine had descended suddenly into a congenitally patent processus vaginalis, and undergone acute strangulation. Also, reasoning from similar cases which I had seen, I expected to find an annular constriction high in the inguinal canal. As eighty-one hours had elapsed since the strangulation had begun, a gloomy view was taken of the condition of the bowel. The operation, which was begun at 9 p.m. on Thursday, July 27th, confirmed our anticipations. The sac contained about two ounces of turbid fluid, mixed with altered blood, and emitting a faint stercoral odour. In the midst of this fluid was a piece of omentum which was almost gangrenous, and a loop of bowel, probably ileum, about six inches long. This bowel was acutely strangulated by an annular construction, so high up the inguinal canal that the aponeurosis of the external oblique and the lower muscular fibres of the internal oblique and transversalis muscles had to be divided to bring it into view and permit the bowel to be inspected at the seat of constriction. The loop of intestine was rough, macerated, and deeply engorged and infiltrated with blood, but not all, I am inclined to think, irrecoverably injured. At the seat of constriction, however, one end of the loop, presumably the proximal, had undergone irreparable injury. Throughout two thirds of its circumference its coats had gangrened and become converted into an ashen grey, bloodless, and friable patch. This formed a crescent, the widest part of which was furthest from the mesentery, and in contact with the constriction. Here there was a small perforation, but elsewhere the peritoneal and areolar coats seemed to be still unbroken.

Although the condition of the patient was critical, I determined to perform enterectomy and immediate suture. Dr. Malcolm ceased the anæsthetic, which he had been administering with great care and judgment, and the

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patient was surrounded with hot-water bottles. The damaged omentum was first removed and the sac thoroughly disinfected, then about four inches of damaged intestine were cut away. The incision through the bowel passed at least half an inch beyond the gangrenous area into healthy tissues, and the whole of the doubtful part of the loop below was also cut away. The escape of fæces from the divided ends was controlled with an elastic band carried round the bowel through a small hole made in the mesentery, and the peritoneal sac was guarded with sponges. None of the mesentery required excision, and the gap left in it by the removal of the bowel was closed with a few points of suture. The ends of the bowel were next united by Czerny-Lembert's method, with an additional row of continuous suture over part of the circumference of the bowel. About twenty interrupted silk sutures were inserted, and an inch of continuous. It required altogether about twenty-five minutes for the excision and suture of the intestine. Afterwards the bowel was disinfected, dusted with iodoform, and returned into the abdomen. Then the whole area of operation was thoroughly sluiced with hot perchloride of mercury lotion (1 part in 1000 parts), and the wound in the abdominal wall, which had been slightly enlarged for the purpose of resection, rapidly closed with a series of deep silk sutures, the lowest of which obliterated the neck of the sac. As the fluid in the sac was obviously septic, a drainage-tube was inserted. During the operation there was considerable shock, but the patient rallied with subcutaneous injections of ether and brandy, and after his return to bed the latter was repeated by Mr. J. Cooper, the house surgeon (to whom I am greatly indebted for his care of this case). During reaction there was some restlessness, for which morphia was given subcutaneously and this was all of that dangerous drug which was given, for I feel sure that it and opium frequently aggravate the intestinal paralysis, which is so frequently fatal in these cases.

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During the reaction the temperature began to rise, and on July 29th, thirty-six hours after the operation, had reached 102·6° F.; it then began to fall, and was normal on July 31st. Flatus was passed the morning after the operation, and a natural action of the bowels occurred on the sixth day. During the first two days the patient was fed by the rectum, afterwards with fluids by the mouth. The drainage-tube was removed on July 31st, and the wound healed rapidly after some altered blood had flowed away. On August 21st he was out of bed, and two days later went to a convalescent home. He was seen four months after the operation quite well, and without any relapse of his hernia.[1]

There can, I think, be no doubt that the bowel in this case was unfit to be returned within the abdomen. Assured of this, I felt no hesitation whatever in excising it. The case was exactly similar to one in which I had seen the gangrenous intestine opened and stitched to the edges of the wound, with a fatal result.[2] Also, further experience has only served to confirm and strengthen the conclusions I arrived at in my previous communication upon this subject. I then argued that the old operation of forming an artificial anus ought to be abandoned because it leaves the patient exposed to the following imminent perils:—(1) non-relief of the intestinal obstruction; (2) spread of the gangrene or ulceration; (3) spread of the septic peritonitis; (4) death from inanition; (5) a further operation for the closure of the artificial anus.

Zeidler, in his recent monograph, to which I have referred, confirms these assertions. He also concludes, as Mr. Kendal Franks and myself had already done, that the mortality after resection and immediate suture is decidedly less (17 per cent.) than that after the older method.

I adopted the Czerny-Lembert suture because it is that

[1] There is now a slight bulging of the upper part of the inguinal canal, but the patient does not care to wear a truss for it. Otherwise he is in perfect health (July, 1894).

[2] "On the Treatment of Strangulated Hernia, &c.," 'Med.-Chir. Trans.,' vol. lxxiv, Case 5, p. 218.

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to which I have become accustomed, and which I can apply most quickly. It has also the advantage of not requiring any unusual or elaborate apparatus. A needle without a cutting edge, such as Hagedorn's, is all that is required, together with the instruments ordinarily used for kelotomy. My own experience of bone plates is as follows. Intending to try them upon a suitable occasion, a bottle-full was obtained from the instrument maker. These dried up and became hard and horny. Others were procured, but they disintegrated and formed a kind of mud at the bottom of the jar. Sometimes, too, when bone plates have been used for immediate suture, especially after gangrene of femoral herniæ, almost insuperable difficulty has been experienced in returning them with the bowel into the abdomen. Cysts have, I believe, also formed in the occluded ends of the bowel beyond the anastomosis. On the other hand, the Czerny-Lembert method has none of these disadvantages, and is safe, provided that plenty of peritoneum is included in the sutures, and provided they are placed in healthy tissues.

Perhaps I may be allowed to refer briefly to the other factors which seemed to conduce to success in this case. One of the chief was the care which was taken to prevent shock, by the cessation of the anæsthetic, the application of warmth and stimulants, and speed in operating. But there can be no doubt that the youth and vitality of the patient aided these efforts. The next was the care which was taken to avoid infection of the peritoneum during the operation, either with the fluid in the sac or with the intestinal contents. But these would have been unavailing were it not that the intestines had not undergone that paralysis which is the most fatal complication of intestinal obstruction. Although not mentioned before, it should be stated that during the operation the small intestines were not very tightly distended, and underwent vigorous peristaltic movements.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. vi, p. 78.)

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ON

PERSISTENCE OF THE THYREOGLOSSAL DUCT,

WITH

REMARKS ON MEDIAN CERVICAL FISTULÆ AND CYSTS DUE TO EMBRYONIC REMNANTS.

BY

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(Communicated by ARTHUR E. DURHAM.)

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AT the present time, judging from the literature of the subject, there is considerable confusion in the matter of fistulæ and cysts in the middle line of the front of the neck. This contribution is made with the hope that these various conditions, arising from the persistence of fœtal or ancestral rudiments, may be more clearly understood. Lannelongue and Achard tend to regard all median cervical fistulæ as branchial in origin. More recently von Kostanecki and Mielecki, and Kanthack have been most dogmatic in their assertions of this as the sole mode of causation.

Several specimens of indubitable persistence of the thyreoglossal duct have come into my hands by the kind-

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ness of the surgeons who treated the cases. These specimens will be described, and an inquiry instituted into the cases which have hitherto been recorded: especially because the influence of von Kostanecki and Mielecki has been such that practically all cases are now huddled together under the heading "Branchial Fistulæ or Cysts."

Cases.

Case I. *Persistent Thyreoglossal Duct; Secondary Fistula; Excision; Relapse.* The specimen about to be described was removed from a well-developed male patient æt. 19, a water-side labourer by occupation.

The history is that a swelling appeared in the middle line close above the sternum, immediately after an attack of scarlatina, when the patient was four years old. This swelling was incised; and ever since there have been relapses of fistula-formation, followed by healing. On admission to Guy's Hospital (on November 27th, 1893) there was a rounded swelling, about 4 cm. in diameter, containing yellow pus, close above the sternum in the middle line; from this a rounded cord could be traced strictly in the median line up to the hyoid bone. The mass was excised by my father as high as the lower border of the hyoid bone, beyond which point the probe would not pass.

The part removed is a somewhat pyriform mass, about 7 cm. long, and tapers from about 3 cm. below to 1 cm. above in breadth and width. The lower portion consists of a mass of ragged granulation-tissue enclosing the cavity which contained the pus; from this cavity a probe can readily be passed up the entire length of the specimen.

The mass presents a groove, better marked anteriorly but traceable posteriorly, which separates it into bilateral portions, a right and a left. On section there is found to be a lumen in each portion: that on the *right* being the one through which the probe passes from the pus-

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containing cavity; that on the *left* apparently ends blindly below, as will be further described. Both lumina are freely open at the upper end of the specimen. The pus-containing cavity is lined by ragged, soft, vascular granulation-tissue, and is irregularly partially divided into a number of loculi by incomplete septa of this material; it possesses no epithelial lining. The lining of granulation tissue extends for some distance up the right lumen, but at the upper part it becomes replaced by a ciliated cylindrical epithelium. The left lumen is also lined with ciliated cylindrical epithelium, except at its upper and lower extremities; at the upper end some squamous epithelium appears; inferiorly its lining ends as a number of solid cords of epithelial cells; here it is surrounded by soft granulation-tissue. A probe passed down this lumen (after hardening) entered one of the loculi of the pus-containing cavity; it would seem probable that a false passage was made leading from the somewhat widened lower extremity of the left lumen.

The bulk of the mass consists of tough lamellæ of fibrous tissue, arranged both longitudinally and circularly; some of the circularly disposed lamellæ form a well-marked septum between the two halves of the specimen. At the upper part this septum, though still well defined, becomes much thinner, and the two lumina approach one another. At the lower part the septum becomes lost in granulation tissue. This tough fibrous tissue contains very numerous bloodvessels. Surrounding the smaller vessels are collections of leucocytic cells. In the present specimen there can be no doubt that they are of inflammatory origin; they merge into the granulation tissue of the pus-containing sac, and also into the sub-epithelial collections of adenoid tissue.

At the upper end of the specimen there are numbers of striated muscle fibres, the majority of which have a longitudinal direction. Many of these are probably fibres of the sternocleidohyoid muscles. Some few, however, are cut very obliquely in transverse sections; a few

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pass into the septum between the lateral parts of the mass (*vide* Plate IV, fig. 1, *mf.*), these apparently belong to the musc. levator corporis thyreoidei.

There are also a few small nerves; it is to be noted that the fistulous track was sensitive.

The upper part of the right lumen and nearly the whole of the left lumen are lined by ciliated epithelium, and give off cœcal diverticula, so that in transverse section there is an irregularly branched cavity more or less surrounded by a varying number of other smaller cavities; the lowest part of the left lumen is a single cavity without surrounding diverticula; at its extremity it gives off a number of processes which end as solid cords of cells. The ciliated epithelium consists of long cylindrical cells with oval nuclei; the nuclei are situated at various levels, leaving a free border of finely granular protoplasm, which is limited by the hyaline refringent border usual in ciliated cells. Each cell possesses numerous cilia (fig. 2).

Besides the shrinkage spaces between juxtaposed cells, there are a few clear spaces due to the presence of goblet cells loaded with mucous material, and provided with nuclei.

Scattered here and there wandercells are seen making their way through the epithelium. One or two of the diverticula are lined by nonciliated mucous cells, cylindrical in shape with peripheral nuclei; in appearance they recall somewhat the cells of the mouths of the gastric glands.

Beneath the epithelium is a variable layer of fine fibrous tissue, for the most part studded with leucocytes. In many places (*vide* fig. 1, *l.*) these leucocytes are so numerous that the tissue becomes like adenoid tissue.

Here and there, where the epithelium is stretched over projecting nodules of adenoid tissue, it becomes much thinned out, or even lost altogether; thus showing the probable history of the lower portion of the right lumen, where only a granulation-tissue lining remains.

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Kanthack considers the adenoid tissue about the foramen cœcum to be of a chronic inflammatory origin, as it is not present in the fœtus or young infant, and is largely increased in cases where the tonsils are enlarged; such, no doubt, is its nature in the present case.

In the sections taken close to the extreme upper end of the specimen there are a few patches of squamous epithelium forming the lining of diverticula of the left lumen (*vide* fig. 3). This is evidence that some part of the ductus lingualis is also persistent. Further evidence is afforded by the recurrence of the fistula, with which the patient left the hospital at his own desire.

He was readmitted on 3rd February, 1894. On February 9th, under an anæsthetic, a probe was passed as far as the lower border of the hyoid bone, and the fistulous track was again excised to that level. An unsuccessful attempt to pass a probe down the ductus lingualis from the mouth was also made.

Three days after the operation stringy mucus exuded from the upper part of the wound. An injection of iodine solution was ordered. On the 18th, when the injection was given, the patient suddenly coughed and said he could taste the fluid, with the sensation of his mouth being "all crinkled up." A few days later an unsuccessful attempt was made with a solution of methyl blue and quinine, in order to test the question of patency, both objectively and subjectively. The patient refused further treatment, and left the hospital. The mucus that exuded was examined microscopically, and found to contain squamous epithelial cells; no cylindrical cells were observed. The portion excised at the second operation was found to be devoid of epithelial lining.

The commencement of the trouble with scarlatina is extremely suggestive that infective material from the faucial region made its way down a patent canal.

It will be remembered that the foramen cœcum is lined by squamous epithelium. I have not been able to discern any transition to the squamous type in the right lumen.

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This specimen differs from those *branchial* fistulæ mentioned by Baumgarten, Neumann, &c., in which the epithelium below is squamous (derived from the sinus præcervicalis of His, and the epiblastic branchial pouch), and above is ciliated; the positions being reversed.

In a series of sections taken near the upper part, in the right side on its mesial aspect (*i. e.* close to the septum), is a small tract of thyroid gland follicles with their colloid contents (*vide* fig. 4). These follicles are not enclosed in a capsule as are the accessory glands, but are intimately associated with a diverticulum, or groove in the right lumen.

Neither at the operation, nor from microscopical examination of snips of the lower parts of the specimen, was any evidence obtained of a connection with the thyroid body, or a lobus pyramidalis. In the lower part of the specimen there are no thyroid gland follicles.

It seems certain that this specimen owes its origin to the *thyreoglossal duct.* This duct, by the researches of His and others, has been shown to bifurcate at its extremity, and the two lumina in this specimen doubtless belong to the forked portion of the duct. The point of bifurcation in this case is above the lower border of the hyoid bone. In Marshall's case[1] it is figured at a lower level. In His's case this point is at the upper part of the thyrohyoid membrane. It would appear from these cases that the locality of the bifurcation is somewhat variable. Besides the anatomical position about the median plane, and the bilateral structure, the presence of thyroid gland follicles is convincing evidence in favour of development from the ductus thyreoglossalis.

The investigations of von Kostanecki and Mielecki, supported by Kanthack, have introduced a further complication in the diagnosis of cysts and fistulæ of the middle line of the neck; namely the possibility of their origin from the *sinus præcervicalis* of His, a cavity which is formed by the overlapping of the hyoid arch in front of

[1] There is some reason to doubt the genuineness of this case, *vide infra.*

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the third and fourth visceral arches, and includes the apertures of the external pouches of the never completed second, third, and fourth branchial clefts. As the sinus præcervicalis extends across the middle line, it is obviously possible that median fistulæ may result from the persistence of its aperture, which normally becomes obliterated (true congenital fistulæ present at birth appear to be exceedingly rare, if indeed there are any really authentic cases); or from the nonobliteration of the included portion, and the secondary formation of a fistula owing to distension of the sac. These authors assert that the only difference that there is between *median* and *lateral* fistulæ is the median position of the aperture of the former.

In median fistulæ it is clear that the diagnosis of the persistence of the sinus præcervicalis can only be made when squamous epithelium occurs about the aperture of the fistula, as this structure is derived from the epidermis, unless a transition from squamous to ciliated epithelium is hypothesised.

It therefore appears to me that several of the cases which von Kostanecki and Mielecki have tabulated as median branchial cysts and fistulæ are in reality of thyreoglossal origin, as will be discussed more fully later.

It is probable that all such cases, in which the lumen passes up in the *mid-line* behind the hyoid bone, and is lined with ciliated epithelium, originate by the persistence of some portion of the ductus thyreoideus or lower part of the ductus thyreoglossalis. Confirmatory evidence may be obtained either by dissection or by microscopical examination; in the former case, connection with a *lobus pyramidalis*, or with the *ductus lingualis*, or with *accessory thyroid bodies*, or by the passage of probes or injected fluid up to or or through the base of the tongue (*foramen cœcum*) in the middle line; in the latter case, by the discovery of a bifurcated condition, or of thyroid gland follicles, or of squamous epithelium in the upper end replacing the ciliated epithelium.

Difficulty arises in cases such as that described by

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Raymond Johnson (Case 1) in which a probe could be passed "more than half an inch above the hyoid bone," the lumen being lined by "a thick layer of stratified epithelium having in parts a distinct papillary arrangement. There are no glands." Kanthack describes numerous racemose glands opening into the ductus lingualis (foramen cœcum). As these mucous glands are abundant just above the hyoid bone, it is reasonable to suppose that they would also be found below it in fistulæ derived from the thyreoglossal duct. Such a condition has yet to be described. In the present state of knowledge it does not appear to be possible to distinguish whether the condition arose from the permanence of the *ductus lingualis*, or of the *sinus præcervicalis* alone, without such minute dissection as could only be undertaken post mortem. The extent to which a persistent sinus præcervicalis may extend up above the hyoid bone is unknown. Hence, though suggestive of such an origin, it does not appear scientific to assign this case definitely to either category.

The presence of squamous epithelium lining the lower part of a fistula must also always suggest the fallacy that it has grown in over bare granulations from the external epidermis, and is therefore quite a secondary condition, having no relation whatever to the transient sinus præcervicalis. In many recorded cases the funnel-like aperture of the fistula was deepened by the traction exerted by movements of the hyoid in swallowing, &c.

Case II. *Persistent Thyreoglossal Duct; Excision; Cure.*—This specimen was removed in 1889, by my father, from a female child six years old.

There was a recalcitrant fistula in the middle line below the hyoid bone. After excision and cauterisation the wound healed well, and the patient has had no further trouble (February, 1894).

Unfortunately the specimen was not carefully hardened for histological study, so that sections are not

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quite so good as could be wished. It is now 1 cm. in length; before shrinkage it was much longer. A probe readily passes through the fistulous track. Microscopical transverse sections at the middle of the specimen show two distinct lumina, each with a differentiated fibrous sheath blending into a common sheath. The connective tissue is less dense and hard than in Case I. One lumen is large, and through it the probe was passed; the other is smaller (·2 mm. × ·05 mm.). Both are lined with the remains of cylindrical epithelium; the preservation of the specimen is not sufficiently good to allow cilia to be seen, whether there were any must be left open to doubt.

Transverse sections at the extreme upper end show the two lumina; [the larger (1·1 mm. × 0·1 mm.) has diminished so that it just occupies the field of a half-inch objective].

Between the two a third cavity can be traced connecting the others.

In all the three lumina the epithelium is partly cylindrical and partly squamous.

There are no circumferential diverticula as in Case I, nor are thyroid follicles seen.

This specimen is to be regarded as a persistent thyreoglossal duct because of its median position, its forked lumen, and the squamous character of the epithelium at the upper extremity.

Case III. *Persistent Thyroid Duct; Isthmian Bronchocele; Excision; Recovery.*—For this specimen, and for the permission to publish it, I am greatly indebted to the unfailing courtesy and kindness of Mr. Jacobson, under whose care the patient was. Nowhere before, I believe, has a similar specimen been described; unfortunately only a small piece is extant, which was preserved and embedded in paraffin by my late brother.

Clinical aspect.—A girl, æt. 18, was under Mr. Jacobson's care for a goître in 1888. The tumour extended from the lower border of the thyroid cartilage to within half

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an inch of the sternum; laterally it passed under the right sternomastoid and reached the anterior border of the left.

At the operation the isthmus alone was found to be affected. "A process running upward on to the thyroid cartilage" had to be cut. The connection with the left lateral lobe was merely fibrous; some healthy gland tissue was removed from the right lateral lobe with the tumour.[1]

The tumour was "almost entirely colloid with cysts of large size." The microscopical sections show several cysts, the largest being about 1 cm. in diameter. The cysts are of three kinds:

1. Cysts lined with *cubical* epithelium, empty or with finely granular contents.

2. Cysts lined with somewhat *cylindrical* epithelium, in two or three layers of cells, which secrete mucoid material. In some of these the secretion has remained discrete, and forms long curved threads of granular material more or less wound up inside the cyst (fig. 5). The condition may be compared to the cylinders of oil paint which exude from the collapsible tubes used by artists. Here and there, there are indications of the presence of cilia, but they are not quite sufficiently well preserved to be made out definitely.

3. Cysts lined with *cylindrical ciliated* epithelium. The epithelium is similar to that described in Case I, so that fig. 2 would accurately represent it, although actually drawn from the other specimen. The contents, where present, are a somewhat coarsely granular material with débris of cells.

The stroma consists of a fine loose reticulum of fibrillæ, with sparse branched and rounded cells. The meshes of the spongework are filled with an infiltration of colloid material, which in some parts forms considerable cell-less masses. In appearance this colloid material resembles the contents of thyroid follicles; but it is loose in the reticulum, and not contained in epithelium-lined spaces, a condition not infrequent in ordinary goîtrous tumours. The data

[1] Before operation the right lobe was thought to be involved.

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for diagnosis in this case consist of (1) the ciliated cystic cavities; (2) the clinical note that at the operation a process was found to run upon the thyroid cartilage; (3) the undoubted connection with the isthmus of the thyroid body.

It does not appear to be possible that the ciliated spaces can be other than unconverted portions of the lower part of the ductus thyreoideus. Whether or no they were connected with a patent canal, extending towards, or actually to the foramen cœcum, must remain unknown.

Speculations as to the association of the persisting rudiment with the colloid infiltration of the isthmus would be at once idle and useless.

This is the only instance I know of, in which there exists pathological enlargement of the isthmus with persistence of the thyroid duct.

Streckeisen describes (Fall 104, p. 234) a universal cystic goître in which there was a ciliated cyst the size of a pea, at the attachment of the musc. hyogl. to the thyroid gland.

REMARKS.

The subject of mid-line cervical cysts and fistulæ is scantily treated of in the text-books on surgery,[1] by English, French, and German authors; and not only is this the case, but the information given seems hardly satisfactory. Such must be my excuse for entering more fully upon this interesting subject—a subject, moreover, which has often been treated with prejudice.

The following classification includes all the possible forms of *cysts and fistulæ of congenital origin in the mid-line of the neck*; the basis is upon embryological grounds.

1. Dermoid Cysts and Fistulæ (epiblastic):
   - A. Those derived from Ductus Lingualis.
   - B. Those derived from Sinus Præcervicalis.
   - C. Those derived from Sinus Præcervicalis with epiblastic portion of gill pouch. (The epiblastic and

[1] The best account is perhaps that of Broca.

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hypoblastic portion of the gill cleft, normally, never communicate in the development of the human embryo.)

D. Those of independent origin, unconnected with any normal embryonic rudiment.

2. MIXED DERMOID AND MUCOID CYSTS AND FISTULÆ (epi- and hypoblastic):

A. Those derived from Ductus Thyreoglossalis.

B. Those derived from Sinus Præcervicalis with Gill-cleft, including the hypoblastic portion.

3. MUCOID CYSTS AND FISTULÆ (hypoblastic):

These, when in the mesobranchial region, that is, limited to the middle line, can owe their origin solely to the Ductus Thyreoideus.

1. DERMOIDS.[1]

A. *Those derived from Ductus Lingualis.*—A very considerable number of cases of dermoid cysts of the floor of the mouth have been collected by Lannelongue and Achard (Chap. 'Kystes Dermoïdes du Plancher Buccal,' p. 230). These are all above the level of the hyoid bone. According to their structure they appear to be of two kinds: (1) those with simple squamous epithelial lining, whose contents are only sebaceous matter; and (2) those in which hair-follicles, hairs, and sebaceous glands, and sometimes papillæ are found. As instances of the former we may cite the cases described by Barker, while an instance of the latter is to be found in Stephen Paget's case, where the lining of the cyst wall is described as "true skin with a few fine short hairs," and some pigmentation—the anatomical attachments not being given. A few cases are recorded in which there was a distinct attachment to the mandible (*e. g.* Ozenne, Obs. 2). It appears, then, that two forms of dermoid cysts occur here, viz. those derived from *ductus lingualis* and those of *independent*

[1] Some authors suggest the term "epidermoids." However, the word "dermoid" is so well understood to mean squamous epithelial-lined cysts, that the refinement appears to be hardly necessary.

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origin. The ductus lingualis is so remote from a region producing hairs, and, moreover, such glands as it possesses being mucous in structure, we should be disposed to rank all dermoid cysts of the floor of the mouth supplied with hairs and sebaceous glands as of *independent origin;* and conversely, at any rate, many of those with simple walls as derived from the *lingual duct.* Unless definable attachment to the hyoid bone or mandible exists, there would appear to be no criterion to distinguish them; except that, in the lingual duct cases, at all events, the genio-hyoglossi muscles are separated, whatever be the state of these muscles in the other conditions (*vide* Ahlfeld's case of ectopia linguæ). Apart from the above remarks on their ontogeny, the full accounts of Ozenne and Barker, and Lannelongue and Achard, render further discussion superfluous here. It is to be noted that they are above the hyoid bone, and that, though usually in the middle line, it is possible for them to assume a somewhat lateralised position by muscle pressure, as Barker has pointed out. This author suggests the sinus præcervicalis as a source; it is uncertain whether this structure can rise above the upper level of the hyoid, probably it never does.

Streckeisen (Fall 102) has found a little dermoid cyst between the hyoid and the upper end of the pyramis thyreoidea, rather in front of, but attached to the latter. It seems impossible to place this in either category (sinus præcervicalis or ductus lingualis).

B & C. *Those derived from Sinus Præcervicalis with or without the Epiblastic Branchial Pouch.*—These are situated below the hyoid bone, and are attached to it. They are lined by squamous epithelium, and are often supplied with hairs and sebaceous glands. Fistulæ appear to be secondary to rupture of a cyst in a large majority of the cases.

Though it is not known at what level the ductus lingualis ceases, the evidence (His) is in favour of its termination being situated above, or at the thyrohyoid membrane.

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Raymond Johnson has described a case of middle-line fistula lined with pavement epithelium, in which a probe could be passed upwards "more than half an inch above the hyoid bone,"—presumably above its lower border. It seems probable, therefore, that this case is to be considered as a persistence of the sinus præcervicalis, rather than of the ductus lingualis. Between sinus præcervicalis dermoids, and independent hypohyoid dermoids there is no diagnostic criterion; the close relation of the sinus to the hyoid bone, to the growth of which it largely owes its existence, would tend to make us regard all examples, with attachment to this bone, as owing their development to this sinus.

The extensive bibliography collected by Lannelongue and Achard concerning median hypohyoid dermoids, suggests that it will be sufficient to notice the few cases in which the branchial rudiment also remained. This is shown by the lateralised passage from a median aperture.

Case 1 (Berkeley Hill).—F., æt. 15. Small sinus opening in mid-line by thyroid cartilage. Probe passes half an inch upwards towards angle of jaw. Initially tumour. No histological examination recorded.

Case 2 (Lefort).—M., æt. 15. Aperture in mid-line at level of hyoid. History of tumour when eleven years old. Track of sinus obliquely upwards, backwards, and to the right towards great cornu of hyoid bone. Excision; persistence of fistula; eventual cure by electrolysis. No histological examination is recorded.

In the absence of knowledge of the microscopical characters it is impossible to assign these definitely here, or under the next heading (Mixed Dermoid and Mucoid).

2. Mixed Dermoid and Mucoid.

A. *Of Thyreoglossal Origin.*—These are more conveniently treated under the next section. For although dermoid lining occurs above, the main part of the aberrant cavity possesses a mucoid lining.

B. *Of Branchial Origin* (lateral track with median aperture).—So far as I am aware, no such case has been

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as yet investigated with the microscope. But it can hardly be doubted that dermoid lining existed in the peripheral part, and mucoid in the proximal part, in the following cases, for the reason that the fistulous track was demonstrated to be pervious into the pharynx. The only cases I know of in which the histology was inquired into, and the mixed condition found, were fistulæ with lateral aperture (*vide* Cusset, Baumgarten, and Neumann).

CASE 1 (Meinel).—F., æt. 34. Median aperture half an inch above sternum. Probe goes upwards and to the right. Pervious to food from pharynx.

CASE 2 (Jenny).—F., æt. 24. Aperture exactly in middle line, opposite lower border of thyroid cartilage. Probe passes up and back to right angle of mandible. Track pervious to pharynx by injections. Tumour first; spontaneous rupture; fistula formed.

CASE 3 (Berg).—M., æt. 21. Aperture in mid-line above incisura cartilaginis thyreoideæ. Probe passes 1 cm. rightward. Pervious to pharynx by injection. Initially tumour. No histological examination.

3. MUCOID.

In the mesobranchial region of His, *i. e.* the median region limited laterally by the gill clefts,—there exist the following structures capable of giving rise to cysts other than dermoids.

1. The acinous glands opening into the ductus lingualis.
2. Remnants of the hypoblastic portion of the thyreoglossal duct—the ductus thyreoideus.

There are a few isolated cases of mucous cysts of the tongue situated in the middle line close to the mucous membrane. Hadden records one, but gives no histological description. There is another specimen in the Guy's Hospital Museum, situated between the position of the foramen cœcum and the epiglottis. Albert also records a case in which there were two cysts in this region. The description, however, is very meagre, and no histological examination is recorded.

A few cases are recorded as accessory thyroid glands

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in the tongue (Rushton Parker, Hickman, Butlin, and Wolf). These have been adversely criticised by Kanthack, who considers them adenomata of tubular glands; and he has reason on his side, for it is impossible to recognise thyroid tissue unless the typical colloid-containing follicles are present. Butlin gives no indication of any such structure in his figure. However, Warren records a case in which typical thyroid gland structure was found microscopically.

Typical accessory thyroid glands ("glandulæ hyoideæ," &c.) have been carefully described, especially by Streckeisen in a most excellent paper (*vide* also Zuckerkandl, Kadyi, and Merten); those that occur within the trachea and larynx (Paltauf) do not concern us here, as they are lateral, and appear to be offshoots from the lateral thyroid rudiments, derived from the region of the fourth branchial bar.

True accessory thyroid glands may occur *above, in front of, behind, below,* and actually *within* the hyoid bone.[1] Similarly, ciliated cysts may occur in the same regions, and many such cases were discovered by Streckeisen in the post-mortem room. Neumann describes an instance simulating a ranula. The examination of a portion of the cyst-wall showed that the lining consisted of ciliated cylindrical epithelium. He recommends removal of a part of the cyst-wall in all cases of suspected ranula for obvious reasons. The rationale of these ciliated cysts is not far to seek. Bochdaleck jun., Krause, Streckeisen, and Rosenberg have found ciliated cells at the lower end of the ductus lingualis, and I can confirm this from personal observation in the post-mortem room. The ciliated cylindrical lining is found both at the termination of the ductus as well as at the openings of the ducts of the mucous acinous glands.

Kanthack was unable to find other than squamous

[1] The terms supra-, infra-, subhyoid, &c., used by some authors are avoidable hybrids; a classical friend tells me there is no objection to the words epihyoid, hypohyoid, retrohyoid, &c.

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epithelium in his researches; this, no doubt, was owing to his imperfect method of only examining microscopically cases in which the presence of a duct was suspected under a dissecting lens. Streckeisen, on the other hand, decalcified the hyoid bone and prepared sagittal microscopical sections of the whole region. In a case that I examined it was impossible to detect the presence of the extremely minute duct in the hyoid region under a dissecting lens; but it became perfectly obvious even to the naked eye upon injection of watery Berlin blue solution from the foramen cœcum.

It is possible for one of these epihyoid mucoid cysts to form a median fistulous track opening above the hyoid bone. Houel records such a case (which von Kostanecki wrongfully quotes as "subhyoid"[1]); microscopical examination revealed the presence of a ciliated epithelium.

Streckeisen and Schlange describe enclosure of the duct by the hyoid bone. Out of the few cases I have examined I found one in which this condition was present.

Dermoid and mucoid cysts may be present together. Ozenne has described one case (Obs. I, p. 281) in which a hair-containing dermoid was found in the epihyoid region, and below and behind it there was a little mucous cyst; he does not give any account of the histology of the latter.

It will be remembered that the ductus thyreoglossus bifurcates at its extremity, that is, the thyroid duct (or hypoblastic portion); indeed by some authors this is considered as an originally paired structure. I have already adverted to the uncertainty of the position of the point of bifurcation. It appears that the two lumina are persistent in the first two cases described above. This condition is either a rare one, or else the cases recorded in the literature have not been thoroughly examined; as indeed is inevitable in those which have been treated by cautery, caustics, or electrolysis. Ribbert's case (*vide infra*) seems to be almost the only case hitherto recorded in

[1] Houel's expression is "sushyoidienne."

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which more than a single lumen exists; he considers it of branchial origin.

The diagnosis of fistulæ and cysts derived from the ductus thyreoideus depends upon—

1. The position strictly in the *middle line*.

2. The presence of *ciliated cylindrical epithelium*, which may be more or less replaced by squamous as the ductus lingualis is approached.

3. Permeability to injections or probes through the *foramen cæcum*; this is rare in the adult, although injections have been successful in the young, interruption of the continuity of the lumen usually taking place at the thyrohyoid membrane owing to movements and tension (His).

4. The presence of *paired lumina*, the remains of the original bifurcation.

5. The presence of *thyroid gland follicles*, or accessory glands intimately associated with the duct.

6. Connection with the *thyroid gland* at the lower extremity.

Of these points the first is absolutely essential. The second is probably almost equally necessary, with the reservation that it is possible that the ductus lingualis (squamous lining) may reach below the hyoid, without persistence of ductus thyreoideus. The other four points are of importance, if present. The presence of a lobus pyramidalis is so common that it would be unsafe to argue from the coincidence unless a given cyst or fistulous track has some essential connection with it.

It is to be noted that the cyst or fistulous cord moves with the hyoid bone on deglutition in nearly all recorded cases—a condition which is a perquisite of thyroid gland tumours according to textbooks on surgery. The hypohyoid dermoids also move with the hyoid bone.

The level of the aperture of a fistula is of no importance. The thyreoglossal duct, descending from the level of the first and second branchial clefts, may have a fistulous aperture anywhere, from a little above the hyoid

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bone to the sternum. Since the early days of the recognition of branchial fistulæ, attempts have been made to mark out the positions upon the neck where the second, third, and fourth clefts will open. It would hardly be worth while referring to the matter here, were it not that such diagrams have been passed on from one text-book to another. A glance at the embryological history of the neck shows how futile such attempts must be. The only means of distinguishing the several clefts is by their internal apertures in the pharynx and by their relations to the nerves, as has been done in a few cases.

The following cases appear to belong, with great probability, to the category of cysts and fistulæ derived from the ductus thyreoideus.

1. Epihyoid Region.—Houel's and Neumann's cases already referred to above, as also those observed by Streckeisen. Delens records many probable cases, but gives no histology (*vide* postscript).

2. Hypohyoid Region.—A. Several cases of median fistulous tracks, with ciliated lining, passing *upwards* have been recorded (Jouon, 1864; Nélaton, 1866; Faucon, 1874; Affre, 1875; Köhler, 1876; Ribbert, 1882; Tillaux, 1885; Arndt, 1881; Strübing, 1892; Schlange, 1893).

All these cases are classified by their authors as branchial fistulæ; Strübing alone suggests the possibility of a thyreoglossal origin. There is no evidence that branchial clefts can exist in the middle line; though, by means of the squamous-lined sinus præcervicalis, they may acquire a median aperture. In Affre's case, there is no possibility of any remnant of sinus præcervicalis, since the exuberant rim of the fistula was shown to be coated with ciliated cells. Ribbert's case appears to be very similar to the one I have described above (Case I). Two tubes were demonstrated by the naked eye; microscopically a third was found between the other two. (It is to be remembered that the ductus thyreoideus is described as breaking up rapidly into a number of anastomosing

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branches, so that the original bifurcation soon becomes imperceptible.) The lumina of these tubes were found to be complicated by folds, and by cæcal diverticula. The lining membrane consisted of cylindrical cells, for the most part ciliated. Squamous epithelium was also present, "especially in the smaller tubes;" but the description is not sufficiently clear as to the exact disposition of this structure.

Arndt regards his case as a palingenesis from the hypobranchial groove of Amphioxus; ciliated epithelium occurs, however, in the thyroid gland of a slightly less remote ancestor, viz. the ammocœte larva of the lamprey. Though embryologists have not described the appearance of cilia in the development of the Mammalian thyroid body[1], yet we may consider ciliated epithelium a normal feature of the ductus thyreoideus when any of it persists at the end of the ductus lingualis (Bochdaleck, &c.).

His and Marshall have described cases as persistent thyreoglossal ducts, their diagnosis being based upon naked-eye anatomy. His made no microscopical examination. Marshall, to whom I am much indebted for showing me his specimens, was unable to obtain sufficiently satisfactory microscopical sections to put his specimen beyond all doubt.

B. Cases of median hypohyoid fistulous tracks or mucous cysts in which no microscopical examination was made are recorded by Dzondi (1829, cases 2 and 3); Riecke (1845), Luschka (1848), Larrey (1852), Chamart (1850), Forget (1852), Hamilton (ten cases) (1870), Bellouard (1875), de St. Germain (1875), Gosselin (1876), Köhler (1876), Cusset (1877), Fischer (1880), Demons (1885), Volkenrath (1888), König (1889), Karewski (1893).

Of these, the series of cysts described by Hamilton are the most interesting; unfortunately he gives no histological account, but as they all contained clear mucoid material, they were all probably of thyroid duct origin, though he

[1] Wölfler figures cells from the teased middle thyroid rudiment of a 15 mm. dog embryo, which appear like ciliated cells, but he gives no description.

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regarded them as bursal in causation, and their contents synovial.

c. Cases in which a median fistulous track led *downwards* towards the sternum. Of these I have been able to collect four, namely, Pooley (*vide* Hamilton) (1870), Roth (1878), Berg (1883), Cusset (1886).

Pooley's case (recorded by Hamilton) is most interesting. Pooley attended a primipara and immediately after delivery the child was noticed to have an abrasion on the front of the skin of the neck, in the median line, over the thyrohyoid space, "as if a scratch with the nail." There was then found to be a little sinus in the mid-line which would admit an "ordinary" probe half an inch downwards and backwards, and a second aperture just above it, one eighth inch deep, as tested by the same appliance. "The openings had the ordinary mucous appearance of such orifices." Whether this track was opened traumatically is left doubtful. The child died when two months old, and no dissection was permitted, nor were any scrapings examined microscopically.

Cusset's case was not examined histologically.

Roth had the opportunity of dissection in a subject with mid-line cervical fistula, down which a probe passed as far as the sternum. Flakes obtained by probe showed both squamous and ciliated cells. Transverse section at middle of the specimen showed cylindrical-celled lining and mucous acinous glands. It was considered as a product of the fourth branchial pouch; and no mention is made of connection to hyoid bone or thyroid body.

Berg found both cylindrical and squamous epithelial cells in the flakes brought away by probing. Examination of the excised track showed a duct, lined with ciliated cylindrical epithelium, which was surrounded by eight or more rounded "acini," also lined with cylindrical epithelium. He believes it to be derived from the fourth gill cleft, "which is not to be doubted when the position and the presence of ciliated epithelium are considered"!—a statement which invites challenge.

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In the light of those cases I have examined, I should regard all these as almost indubitable instances of persistent ductus thyreoglossus. For it is extremely difficult to imagine how a branchial cyst could come to occupy such a position; whilst such a ciliated cyst as Streckeisen found attached to the thyroid isthmus, or Case III recorded above, if it happened to rupture, could easily give rise to a similar condition.

Prognosis and Treatment of Thyroid Duct Persistence.

Whilst it is true that some recorded cases of cyst have disappeared *suâ sponte*, yet as a general rule the prognosis and treatment are interdependent. It must also be remembered that a mere puncture of one of these cysts may give rise to a troublesome fistula; for instance (Larrey's case), a soldier was troubled with the pressure and friction of his tunic against the "tumour," so he pricked it with a needle. After this he had a secreting fistula, for four years under surgical treatment; it was eventually cured by iodine injections.

Cases have been treated by incision, injection, caustics, actual cautery, electrolysis, and excision; in some, success attended the treatment more or less immediately; in others the patient was lost sight of—with his fistula. In some recorded cases the perseverance both of the patient and of the surgeon is remarkable. Thus Gosselin describes a case which was severally treated by incision and excision; then nearly all the known caustics were tried in succession, at intervals of some few weeks for a period of two years. The patient still had the fistula patent, and M. Gosselin was still hopeful that caustics would eventually cure him. It is obvious that such unsuccessful attempts are due to incompleteness of the interference with the epithelial-lined track, the reason whereof is the narrowness of the lumen as it passes behind the hyoid bone, or indeed actually perforates that bone. Recognising this fact, Schlange has cut through the hyoid bone and by "forcibly retracting the two halves,"

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obtained a good view and room enough to deal with the tissues in the middle line far up towards the root of the tongue. He states definitely that no trouble was experienced from this procedure, either at the operation or after it. Indeed, in one case he removed a piece one centimetre broad from the middle of the hyoid bone. The extraordinary obstinacy of some of these fistulæ in the past, together with the sound basis of anatomical conditions, as well as the absence of disagreeable results (immediate or remote), make it probable that section or resection of the hyoid bone will be resorted to without compunction in the future, when less complete measures have failed.

From attempts to trace the ductus lingualis in the post-mortem room, I should be inclined to think that the injection of a saturated solution of Berlin blue into the fistula would be useful before proceeding to excise. The blue precipitates and leaves an unprobeable and otherwise invisible canal perfectly obvious as a thin dark blue line. By this method it would be easy to discern whether the upper limit of the fistula had been reached.[1]

FURTHER REPUTED ORIGINS OF MEDIAN CYSTS AND FISTULÆ.

Besides the above conditions, the following other reputed causes of median cysts and fistulæ remain to be shortly considered.

1. *Enlargement or Cystic Degeneration of Præepiglottic Mucous glands* (Nélaton). It has been adversely criticised that the thyrohyoid membrane must have become perforated for these to present below the hyoid bone. There is no evidence in favour of this view, except in so far as that the "glands in front of the epiglottis" and the glands opening into the lingual duct are identical.

2. So-called *Tracheal Herniæ*. Several cases have been collected by Eldridge; none of them appear to be entirely

[1] Any excess of the blue can be removed by a weak alkaline solution.

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satisfactory—moreover air has been blown through fistulæ, *e. g.* Fischer's case. The common paired condition suggests ductus thyreoideus, or bilateral persistence of branchial pouches as the real condition. Until a dissection has been performed these tracheal herniæ must be considered doubtful.

3. *Enlarged Bursæ about the hyoid bone, thyroid cartilage, and geniohyoglossi.*

Several bursæ are described in these situations, and these have been pronounced guilty of cyst-, and fistula-formation.

Verneuil's classification is the one most frequently adopted, and which has crept into most text-books, English and foreign, viz.

1. *Bursa mucosa infrahyoidea seu thyreohyoidea* (Bourse séreuse de Boyer).
2. *Bursa mucosa suprahyoidea.*
3. *Bursa mucosa thyreoidea.*

Notwithstanding the common belief that these structures are the causes of median-line cysts and fistulæ, I have been unable to find a report of a single authentic case.[1] The merits of the belief are well characterised by Verneuil's statement in 1877, at the discussion on Houel's case, "on devra s'appuyer comme caractère fondamental sur l'épithelium à cils vibratiles qui les tapissent; alors que les autres (fistules branchiales) ne presentent que de l'épithelium pavimenteux." A bursal cyst with ciliated lining would indeed be a prodigy!

In conclusion, it is hoped that the evidence given that the thyreoglossal duct may persist is sufficiently convincing to prevent the reappearance of any such denials as have been published in the past.

The characters of three cases have been described, and

[1] The case recorded by Verneuil of epihyoid "bursal" cyst (op. cit. infra, p. 192) is hardly satisfactory, as the absence of epithelium attached to the cyst-wall is amply accounted for by the macerated condition of the tissues of dissecting-room subjects.

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the means whereby they may be identified have been pointed out. It is suggested that many of the cases of "*median branchial cervical fistula*" which have been published are in reality cases of *persistent ductus thyreoglossus*.

The thorough examination of all specimens met with in the future is much to be desired; more especially to determine whether bursal enlargements, which have been described about the hyoid bone, are not pathological creatures of the imagination.

Postscript.—Since the above paper was written, Dr. Buscarlet has kindly sent me a copy of his thesis. He records (1) a case of median epihyoid fistula lined with ciliated epithelium (*vide* pp. 215 and 217 *supra*). (2) A case of median hypohyoid fistula lined similarly. (3) A case of epihyoid dermoid fistula lined with squamous epithelium (*supra*, p. 210).

All three appear to be derived from the ductus thyreoglossus; the former two from the ductus thyreoideus, the latter from the ductus lingualis. It is therefore hardly desirable to call them "Fistules Branchiales," since the author admits their origin from the "Canal de Bochdaleck" or thyreoglossal duct of His.

In each case operative interference was practised more than once, owing to recurrence of the fistula.

Messrs. Whitehead, Hardie, and Morton have each recorded cases of mucoid fistula in the median hypohyoid region. In one case (Hardie's) ciliated epithelium was found; the other cases were not examined microscopically. I have to thank these authors for kindly replying to my enquiries.

The specimens obtained in Cases I and II have been presented to the Museum of the Royal College of Surgeons.

Case I formed the basis of a clinical lecture by my father; this is reported in the 'Clinical Journal,' vol. iii, 1894, p. 150, and in the 'Guy's Hospital Gazette.'

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(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. vi, p. 83.)

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DESCRIPTION OF PLATE IV,

On Persistence of the Thyreoglossal Duct; with Remarks on Median Cervical Fistulæ and Cysts due to Embryonic Remnants (HERBERT E. DURHAM).

FIG. 1.—Transverse section (× 8·4) at the upper end of the specimen removed from Case 1. (3 in. obj. cam. luc.). On the right side (R) are seen three cavities representing the right lumen and two diverticula. On the left side (L) there are several cavities representing the left lumen and its diverticula. *art.* Arteries. *cd.* Débris of cells and mucus. *d.* Septum between the right and left sides. F P. False passage made for purposes of orientation. *l.* Lymphoid or adenoid tissue, represented diagrammatically with fine dots. *mf.* Striated muscle fibres. *s.* Situation of the thyroid follicles.

FIG. 2.—Ciliated cylindrical epithelium from one of the duct cavities. ($\frac{1}{6}$ in. obj. cam. luc.)

FIG. 3.—Squamous epithelium from one of the diverticula of the left lumen. ($\frac{1}{6}$ in. obj. cam. luc.) *ct.* Connective tissue.

FIG. 4.—A thyroid follicle from the neighbourhood of the right lumen. The colloid contents show the hemispherical depressions commonly seen in sections of the thyroid body. ($\frac{1}{2}$ in. obj. cam. luc.)

FIG. 5.—Portion of cyst in Case 3, showing the threads of secretion (*m.*) attached to the epithelial cells; also the hyaline interruption between the granular thread and the surface of the cells (*ep.*). The loose reticular tissue (*rt.*) with colloid infiltration (*c.*) is seen below. (1 in. obj. cam. luc.)

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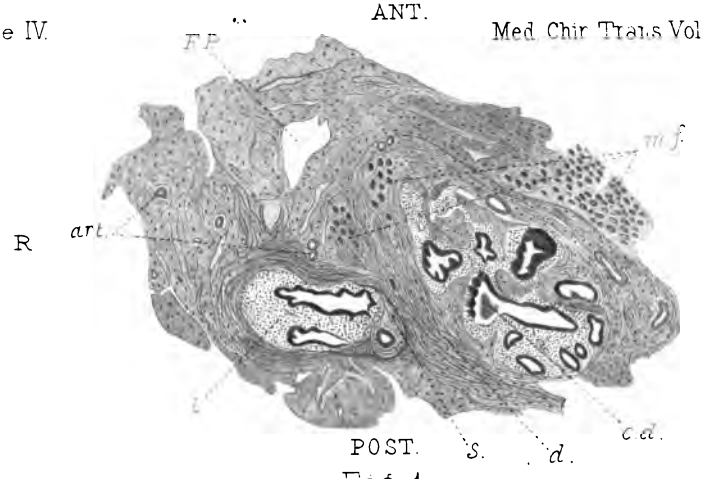

Fig. 1.

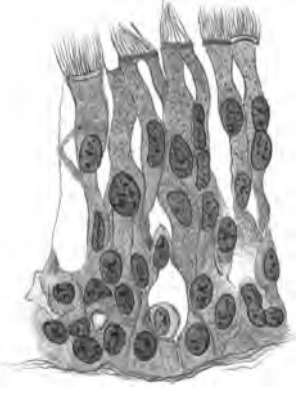

Fig. 2.

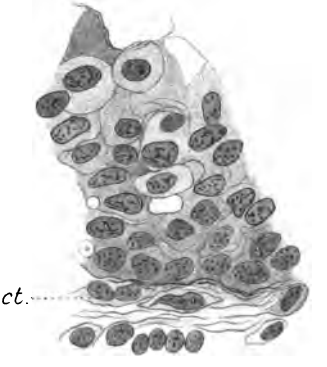

Fig. 3.

Fig. 4.

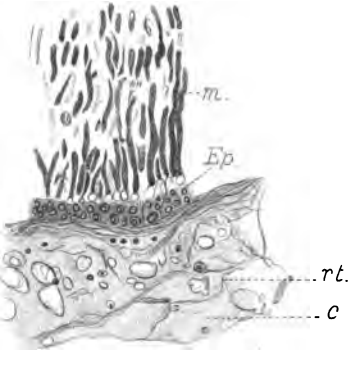

Fig. 5.

H.E.D. del.

Danielsson & Co. sculp.

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A CASE

OF

EXTRA-PERITONEAL VESICAL HERNIA.

BY

ERNST MICHELS, M.D. (BERLIN), F.R.C.S.ENG.,
SURGEON TO THE GERMAN HOSPITAL.

COMMUNICATED BY MR. R. W. PARKER.

Received March 5th—Read April 24th, 1894.

THE presence of the *bladder* in a hernial sac has been looked upon as a matter of pathological rather than practical surgical interest. But in these days, when the radical cure of hernia is so frequently undertaken, it may be expected that the surgeon will more frequently meet with the rarer forms of hernia, and that he may find difficulties in recognising them and dealing with them. I am not aware of any case published in this country in which the presence of the bladder in a hernial sac led to complications during an operation; and I think, therefore, the following brief notes will be of some interest.

H. K—, æt. 48 years, was admitted into the German Hospital on September 18th, 1893, under the care of my colleague Dr. Port, suffering from an intercostal neuralgia consequent on an old fracture of the ribs. The patient

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had inguinal hernia on both sides, which caused him constant annoyance, and as a truss gave him much pain, and could only be worn at intervals, the patient assented readily enough when the radical operation was suggested to him. Dr. Port kindly transferred the case to me, and on September 30th a radical operation was successfully done on the left side, according to Banks's method.

On October 28th the right hernia was operated upon; it was only small—of the size of a walnut—and apparently quite reducible. After reduction some thickening remained, which was thought to be due to the hernial sac. A longitudinal incision was made in the long axis of the hernia, and the external layers were cut through; the parts were much matted together, and it was quite impossible to distinguish between the layers: among them some bundles of interlacing muscular tissue attracted attention, but their exact significance was not made out, as the matting of the parts had effaced all anatomical landmarks. At last what appeared to be the sac was reached, opened, and found empty, only a little clear straw-coloured fluid escaped; the apparent sac was isolated from the surrounding structures, which was effected with some difficulty, owing to its dense adhesions and to its extraordinary softness and friability; it was then tied at its neck, and the stump was returned into the abdomen. The walls of the inguinal canal were brought together by four deep sutures.

We were quite aware after the operation that we had to do with a most unusual case; the anomalous layer of muscular tissue, the great softness and aptness to tear of the sac, the escape of fluid from an otherwise empty sac, were points which deserved and attracted our attention, but we failed to come to a correct interpretation until twenty-four hours afterwards.

At that time, after having been in an entirely satisfactory condition, the patient became restless, complained of sickness and of pain in the hypogastrium, and the urine he passed contained a large admixture of blood. I saw

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him a few hours later, and now the unusual conditions which had remained unexplained at the time of the operation became perfectly intelligible. What I had taken for and treated as the hernial sac was, in reality, a diverticulum of the bladder which had descended through the inguinal canal, and had become adherent to the neighbouring tissues. The muscular bundles belonged to the bladder, and the clear fluid which had come away during the operation, and which we had believed to be peritoneal fluid (without inquiring sufficiently into its character), was urine. No hernial sac, in fact, had existed, and we had to do with one of the most unusual conditions in surgery, namely, hernia—or more correctly (since there was no sac) descent—of the bladder through the inguinal canal.

The question now arose how to repair the damage I had done. I thought it best to expose the injured bladder as fully as possible, to ascertain the extent of damage, and then to act accordingly. I opened the wound, removed the sutures closing the inguinal canal—the walls of which thirty hours after the operation showed distinct signs of commencing adhesion,—and, as this did not allow of a sufficiently free view, I prolonged the incision upwards and opened the abdominal cavity. With the help of a silver catheter and injection into the bladder, I was able to make out a wound a little more than one inch long in the extra-peritoneal part of the bladder. The bladder itself was considerably enlarged. A double row of sutures (avoiding the mucous membrane) was inserted until the wound was closed and watertight; the peritoneum and the upper part of the abdominal wound were closed, but the lower part of the wound was left open and plugged with iodoform gauze, to prevent infiltration of urine in case the sutures in the bladder should give way. A Jacques catheter was passed into the bladder and left there for six days; as long as it was retained the bladder was washed out twice a day with boracic lotion, carefully avoiding, of course, over-distension. On the following day the temperature rose to 101·2° F., but

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from that date the patient made an uninterrupted recovery; the urine became normal after thirty-six hours. As the lower part of the wound was allowed to heal by granulation, its closure took up a considerable time. The patient was discharged early in January with his wound completely healed, and quite free from any pain or discomfort in micturition. He was advised to wear a truss permanently, lest the healing of a comparatively large wound by granulation should tend to weaken the abdominal wall.

Continental surgeons have more frequently met with similar complications, and lately Iovars in the 'Revue de Chirurgie' has collected a number of interesting cases; other papers have been published by Mandry (Tübingen), Lanz (Berne), Roth (Berlin), and Reverdin (Geneva). I do not propose to enter into all the more or less interesting details of the cases, but will only call attention to some of the principal points.

It appears that we have to distinguish two entirely different forms of vesical hernia. The anatomical characteristics of the first and much more common form seem to be the following: an ordinary hernial sac descends through one of the hernial canals; it is found either empty or containing some abdominal organ; in descending, the sac has inverted and taken down through the canal a part of the intra-peritoneal portion of the bladder, which is now found behind the sac. If an operation either for radical cure or for relief of strangulation is done, after reduction of any contents of the sac which may be present, another structure is found behind the sac to which it is generally very closely adherent. In the great majority of recorded cases this obscure structure has been injured either inadvertently in the attempt to separate it from the sac, or intentionally in the belief that a second sac was being dealt with. Even then, only in a part of the cases was a correct diagnosis made and the proper treatment—suture of the bladder—adopted; in other cases the apparent second sac was ligatured at its neck, and an urinary fistula followed which closed spontaneously after

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some time. Reverdin had a case of this kind; after the operation he had his suspicions, and microscopical examination showed that what he had removed as a hernial sac was in reality the mucous membrane of the bladder.

Some surgeons, however, have been successful enough to avoid injury to the bladder in these cases. Roth, Boekel, Lanz, who had the good fortune to meet with two or more cases of this kind, were enabled, after having cut into the bladder in their first cases, to profit by the experience so gained, and to avoid injury to the bladder in the subsequent cases. Krönlein cut through the serous covering of the viscus, but had his suspicions aroused by the well-marked muscular layer, which put him on his guard, and enabled him to recognise the organ he was dealing with. Kummer and Walter, who also found the bladder behind an omental hernia, were able to ascertain the real state of things by enlarging the ingninal canal, passing the finger into the abdomen, and tracing the connections of the structure before them.

The cases of the second variety—descent of the extra-peritoneal part of the bladder through a hernial ring—of which my case is an example, are much less frequent. They are, nevertheless, of great surgical interest, not only since injury to the bladder seems unavoidable, but also because in most cases the real state of things was not discovered till some time after the operation. I have found five such cases recorded.

In all these cases the diverticulum of the bladder was thought to be an empty sac, and was excised; in Pilz's case the diverticulum had become gangrenous in consequence of very tight constriction. Polaillon and Postemski recognised the true state of things by the flow of urine from the apparent sac, and closed the wound of the bladder by sutures. Aue stitched what he believed to be the empty sac to the skin; the next day the dressing was soaked by urine. Siegel's case (Würzburg) is exactly like my own; symptoms of extravasation of urine and hæmaturia indicated the injury to the bladder, and laparotomy and subsequent

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suture of the bladder (two days after the injury) saved the patient's life.

In conclusion, I think the following points are of practical clinical interest:

1. It is almost impossible to make the diagnosis of vesical hernia previously to operation. One case only is recorded (from Buenos Ayres) where the clinical symptoms pointed to the presence of the bladder in the hernia; in Pilz's case the signs of urinary trouble were due more to the long-standing cystitis and pyelitis than to the hernia. In all other cases the bladder was found quite unexpectedly.

2. It is extremely difficult to avoid injury to the bladder. If, after opening the sac and reducing the contents, another sac-like structure is found behind the first sac and adherent to it, it seems the best proceeding to enlarge the inguinal ring, pass the finger into the abdomen, and try to make out the nature of the case in this way. In those rare cases where the extra-peritoneal part of the bladder descends through the inguinal canal perhaps the peculiarly arranged muscular bundles may be detected and recognised; but even this sign will fail in cases where a diverticulum of the mucous membrane passes through a gap in the muscular coat: in any case, if suspicion has been once aroused, it is easily verified by the introduction of a silver catheter, and better still by the injection of fluid into the bladder.

3. The prognosis in these cases is fairly good, only two fatal cases being recorded: in one of them (Pilz) death was due to the pre-existing cystitis and pyelitis; in the second, shock was the cause of death (Polaillon). All the other cases recovered, some of them after formation and spontaneous closure of an urinary fistula.

4. If the bladder has been wounded, it should be sufficiently exposed as soon as the nature of the injury is clear; and the wound closed by suture. This seems almost needless to say, but it is only fair to mention that a number of cases have recovered without suture, generally

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after having gone through the stage of an urinary fistula. Reverdin, for instance, trusted to the ligature he had tied round the neck of the diverticulum, which he hoped would hold till adhesions had shut off the peritoneal cavity; he was quite successful in his case, but he thinks himself that full exposure of the wound and closure by sutures would have been the safer and therefore preferable treatment.

The majority of the cases published up to the middle of 1892 have been collected and tabulated by Iovars, 'Revue de Chirurgie,' 1893, 1, 2.

More recent cases and additional references will be found in the following papers:

Mandry, 'Beiträge zur klinischen Chirurgie,' 1893.

Roth, 'Deutsche medicinische Wochenschrift,' 1892, No. 23.

Laur, 'Berliner klinische Wochenschrift,' 1892, Nos. 30, 31.

Of special interest are—

Reverdin, 'Revue médicale de la Suisse romande,' 1890, p. 698.

Pilz, 'Wiener klinische Wochenschrift,' 1891, No. 19.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. vi, p. 89.)

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AN OPERATION

FOR THE

CURE OF CLEFT OF THE HARD AND SOFT PALATE.

BY

N. DAVIES-COLLEY, M.C.,

SURGEON TO GUY'S HOSPITAL.

Received April 19th—Read April 24th, 1894.

For many years past I have been in the habit of operating in some cases of cleft palate by carrying a triangular flap cut from one side of the hard palate across the cleft, and attaching it to the other side. More than three years ago I published a description of my method in the 'British Medical Journal,' and I then stated that my operation was applicable (1) in infants, (2) in cases in which the ordinary operation had failed, and (3) in cases in which the cleft of the hard palate was too wide to be bridged over by the ordinary operation.

Last year it occurred to me to modify and extend my original operation; and the success which I have obtained in the six cases in which I have adopted my new method emboldens me to bring it before your Society, and to recommend it as applicable to all cases of cleft of the hard and soft palate.

The method is somewhat complicated, but I think that by the help of diagrams I can make my description of it generally intelligible. It may be divided into three stages.

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First Stage: that of Incision and Separation of the Muco-periosteum.

The patient being under an anæsthetic, and the jaws held open by Smith's gag:

(*a*) An incision, *a b*, is made from before backwards, about an inch long, with its centre just internal to the last molar tooth. It should go down to the bone in front, and behind it should pierce the soft palate. Through this incision a raspatory is introduced, and the soft parts separated inwards from the posterior half of the hard palate, much as in the ordinary operation, but not to such an extent.

(*b*) An incision, *c d*, is carried on the same side from just in front of the cleft, and at a distance of about a quarter of an inch from its margin, backwards to the junction of the hard and soft palate. As it approaches the soft palate the incision should converge to the edge of the cleft, and it should be continued along the edge of the soft palate in such a way as to split that structure to the depth of about three eighths of an inch. For this purpose the knife should be lateralised, and as the knife approaches the uvula a forceps will be required to hold the uvula steady while it is being divided. At this part the incision must be not quite so deep, in order to avoid the complete division of the lateral half of the uvula. The soft palate near the cleft will now consist of two planes,—a lower one which is continuous with the band of muco-periosteum between the two incisions, *a b* and *c d*, and an upper one attached to the back of the hard palate. The muco-periosteum internal to the incision, *c d*, should be separated *inwards* from the bone until it is left attached by the soft tissue which covers the margin of the cleft of the hard palate.

(*c*) A large flap, *e f g*, of somewhat triangular shape, but with the front angle rounded, should be taken from the other side of the palate.

One side of the flap, *e f*, runs parallel to and a sixth of

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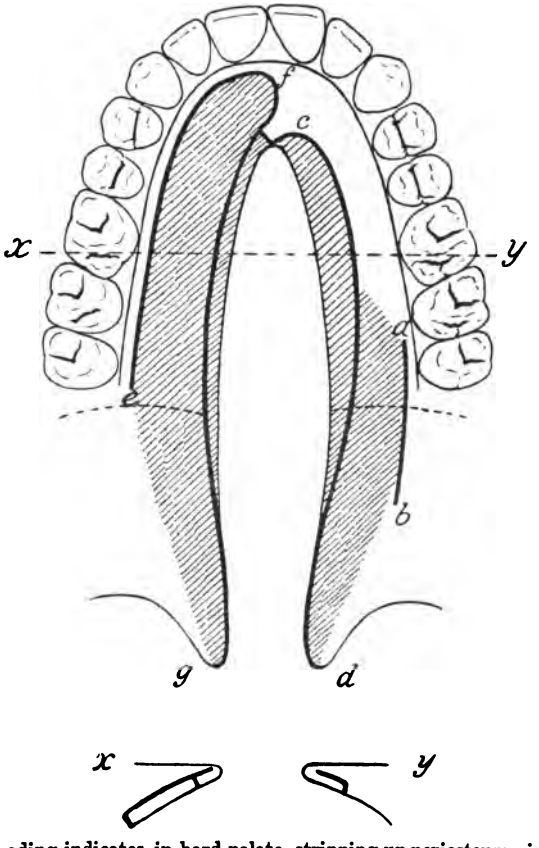
an inch from the insertions of the teeth from the last molar to the median incisor; the other, *f g*, runs backwards at a distance of a sixth of an inch from the margin

FIG. 1.



Shading indicates, in hard palate, stripping up periosteum; in soft palate, splitting into two planes. The lower figure represents diagrammatically a transverse vertical section through the hard palate along the line *x y* in the upper figure.

of the cleft of the hard palate, and is continuous with a splitting of the soft palate similar to that upon the other side, and reaching as far back as the tip of the uvula. The muco-periosteum of the triangular flap should also be separated from the hard palate by means of the ras-

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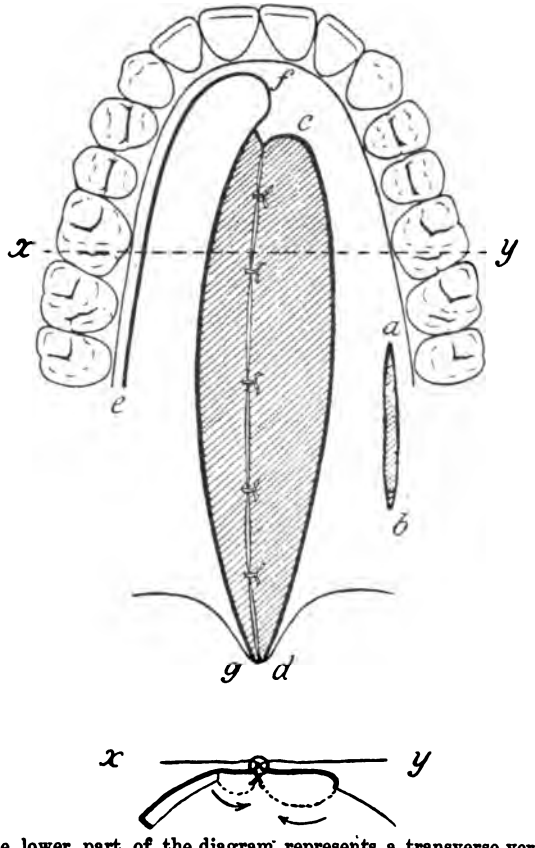
patory, and finally that which lies internal to *f g* should be separated inwards, until it is only attached to the margin of the cleft.

Second Stage: the Union of the Mesial Flaps and the Upper Planes of the Soft Palate.

By means of an ordinary palate needle, with the curve at the end in a plane at right angles to the stem, fine

FIG. 2.



The lower part of the diagram represents a transverse vertical section through the hard palate along the line *x y*, in the upper part of the diagram. The arrows indicate the direction in which the muco-periosteum of the margins of the cleft is reflected inwards.

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sutures of silk or catgut are passed through the edges of the flaps internal to *c d* and *f g*, care being taken to turn the flaps inwards, so that their mucous covering looks upwards, and their raw surface downwards. Continuously with this union the edges of the upper plane of the soft palate on either side must be brought together in the same way. From four to six sutures are necessary for this stage. When it is completed the whole cleft of the hard and soft palate should be bridged over by a layer of muco-periosteum and soft palate tissue, with the raw surface looking downwards.

Third Stage: Union of the Triangular Flap and the Lower Planes of the Soft Palate.

With the same needle sutures of soft silver wire are now passed in the usual way, so as to draw over the margin *f g* of the triangular flap to the outer edge of the incision *c d* on the other side. At the same time the margins of the lower planes of the split soft palate are brought together in the same way. About six wire sutures are necessary, and two silk or horsehair sutures may be used for the approximation of the uvula and the adjacent parts of the soft palate. There will now be a second complete bridge across the cleft, but in this bridge the mucous surface will look downwards, while the raw surface will look upwards, and be in contact with the raw surface of the first bridge. The incision *a b* gapes widely, and may have to be increased in size, especially at the expense of the muscular tissue of the soft palate, in order to allow the edges to come together without tension.

The after treatment is similar to that which is usual after the ordinary operation. I have generally allowed an interval of at least three weeks, and sometimes as many as six weeks, before removing the sutures of the third stage, while those of the second stage have to be left to come away as they can, or to be absorbed.

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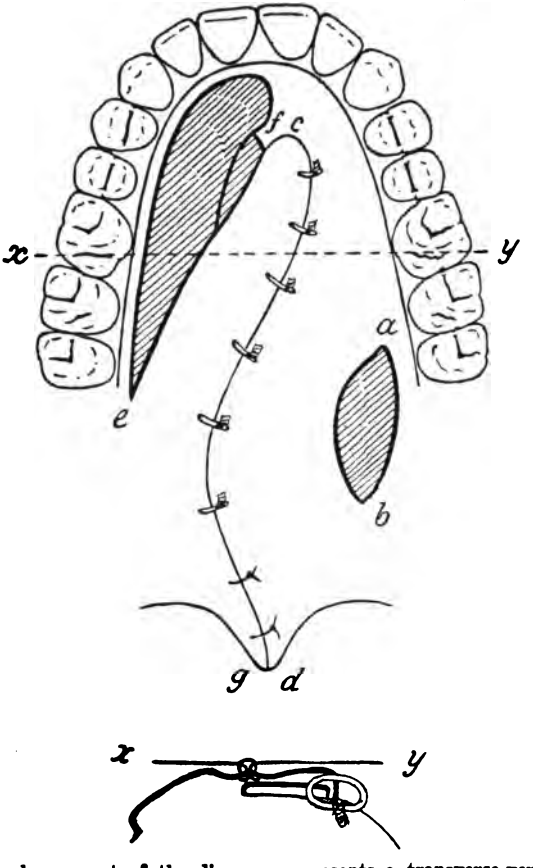
There is so little tension, that if primary union should fail, secondary union would probably take its place. For a short time a raw surface is left in the opening made by

FIG. 3.



The lower part of the diagram represents a transverse vertical section through the hard palate along the line *x y* in the upper part of the diagram.

the gaping of the incision *a b*, and on the other side over the space previously covered by the triangular flap; but these surfaces soon get covered with granulations, and give rise to no trouble or deformity.

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The advantages I claim for this method of operation are—

1. No tissue has to be pared away.

2. A much larger extent of raw surface is brought into close contact than in the ordinary operation.

3. The tension, at any rate in the lower bridge uniting the sides of the hard palate, is much less.

4. Whereas in the ordinary operation the pressure of the tongue tends to tear apart the slender line of raw surface which has to unite, in this operation the pressure is beneficial, as it keeps the raw surfaces of the two bridges in close contact.

5. In cases in which only a part of the hard palate is cleft, some advantage is gained by using the muco-periosteum from one side of the undivided part of the hard palate to make good the deficiency in the posterior or divided part.

No special instruments are required for the operation, but I have found a metal ring at the end of a stem of considerable help in passing sutures. This ring is held at one side of the muco-periosteal flap, while the needle is thrust through from the other side. I have also occasionally used a double-edged scalpel, with the blade at an angle with the handle, for splitting the edge of the cleft soft palate.

The disadvantages which occur to me are—

(1) The operation takes about half as long again as the ordinary operation, because of the number of sutures to be inserted.

(2) In some of my earlier cases, in which I operated on the hard palate only, a small gap was left in the front of the bridge.

This, however, has not been the case lately; and if it should occur, it could be easily remedied by carrying a triangular flap across from the other side in a subsequent operation.

It may perhaps be thought that there is danger of sloughing when so large a flap has been so freely detached. I have never known this to occur; I suppose because

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there is such a free supply of blood to the base of the flap from the posterior palatine artery. As yet I have not had the opportunity of operating upon an infant by this method; but I have had such good success in uniting the hard palate by my flap operation in infants as young as fourteen months, that I shall have great confidence in adopting my new method even at that early age.

In conclusion, I append a short account of the six cases upon which I have already operated in this way.

I think the success obtained will bear comparison with any which has followed a similar series of cases operated on by the ordinary method. At any rate, it is much greater than the success which I have ever obtained by any other method of operation.

Appendix to Paper describing a Mode of operating upon Cleft of the Hard and Soft Palate, by N. Davies-Colley.

Case 1.—Samuel C—, æt. 21. Congenital cleft of the whole of the soft and half the hard palate. Operation under ether, May 19th, 1893.

On June 14th there was complete union, with the exception of a narrow gap, a quarter of an inch long, at the anterior extremity of the cleft.

Case 2.—Hilda M. T—, æt. 6. Congenital cleft of all the soft and three quarters of the hard palate. Cleft in front half an inch wide, behind three quarters of an inch. Operation July 15th, 1893.

September 22nd.—Sutures removed, union good. The next day the child had scarlet fever.

December 1st, 1894.—Union complete with the exception of an opening one eighth of an inch in diameter at the junction of the hard and soft palate.

Case 3.—Walter P—, æt. 20. Cleft of all the soft palate and a small part of the hard palate. Operation September 26th, 1893, under chloroform. All united except about half of the uvula, which remained bifid.

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Case 4.—Mostyn B—, æt. 8. Complete cleft of the hard and soft palate, but with the two portions of the alveolar process in apposition. Operation October 24th, 1893.

December 14th.—Good union, with the exception of the posterior half of the soft palate.

Case 5.—Helen C. M—, æt. 9. Cleft of all the soft and half an inch of the hard palate. She had been operated upon by the ordinary method at another hospital three months before. The left side of the soft palate was shorter than the right, and the left half of the uvula somewhat small and contracted. Operation under chloroform January 10th, 1894.

February 26th.—Good union, with the exception that the left half of the uvula has disappeared. She can talk better.

Case 6.—Henry H—, æt. 15. Double harelip with complete cleft of the hard and soft palate, the cleft extending forwards between the premaxillary bone and the alveolar process of the left maxilla. Operation under chloroform January 26th, 1894.

February 22nd.—All the sutures were removed. Union complete, with the exception of the alveolar process. The union extended forwards to half an inch behind the insertion of the left canine tooth.

28th.—The double harelip was successfully operated on. It is probable that the pressure of the upper lip will in time bring the premaxillary bone into proper relation with the alveolar process of the left maxilla, and so close the remainder of the cleft.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. vi, p. 94.)

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A CASE

OF

EXTREME PROLAPSE OF THE FEMALE URETHRA

IN A CHILD AGED SIX.

BY

THOMAS BRYANT, M.Ch., F.R.C.S.,

CONSULTING SURGEON TO GUY'S HOSPITAL.

Received March 6th—Read May 8th, 1894.

On June 14th, 1893, I was asked by Dr. A. Atkinson to see Miss B—, aged six years, for what, on examination, proved to be a complete prolapse of the urethra. The child was not apparently in much suffering, but passed urine somewhat frequently. On examination, the vulva and the parts about were found bathed with a blood-stained mucus, and the everted urethra appeared as a rounded swelling nearly one inch in diameter, of a deep purple colour from congestion, and projecting about three quarters of an inch from the soft parts beneath (*vide* Plate V). Upon the surface of this swelling there was a central conical orifice, through which a catheter readily passed into the bladder. The urine drawn off was quite clear.

From the history of the case it appeared that, at the age

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of three, after some genital irritation, the child's linen was stained with blood, but as this condition soon passed away with the local irritation, no examination of the child was made, and consequently no opinion as to the cause of her symptoms was formed. In the course of the next three years, three or four other similar attacks occurred, but each one passed off naturally in a few days.

The present attack came on five days before I saw her, with local genital irritation and straining, which, being supposed by the mother to be connected with the bowel, was treated as such by her, but not professionally dealt with.

On the third day, as the symptoms had steadily increased in severity and the discharge of blood-stained mucus became more copious, Dr. Atkinson was called in, and as he found the nature of the case was not very clear, he suggested that I should see it with him. When I first saw the child I confess to having been somewhat puzzled, as I had neither seen anything like it nor had I read of such a case. What I was looking at was a cherry-red, blood-oozing, projecting mass, and was clearly a prolapsed urethra, as indicated by the plicated folds of mucous membrane about a central depression upon the surface of the protrusion, and by the fact that this central depression proved to be the orifice of the urethra. The cause of the prolapse was, however, obscure. The question of its being a venous nævus was entertained but dismissed, as was the possibility of its being a urethral caruncle; for neither nævus nor caruncle could assume the appearances which this case presented.

The view I was inclined to take was that there existed some local irritation at the neck of the bladder, which had led to its severe expulsive efforts, and hence the prolapse; and in this view Dr. Atkinson coincided.

With this "working diagnosis" it was therefore arranged to give the child an anæsthetic and explore the bladder through the urethra by the introduction of the finger, the introduction of the finger being regarded as

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the best means of reducing the prolapse. This was done on the 15th of June.

The introduction of my little finger through the urethra into the bladder was easily effected, after the passage of two of my small conical ivory urethral dilators; but the result of the examination was negative, for I failed to find any evidence of bladder trouble, or indeed of any other urethral trouble, than its prolapse. I consequently completed the reduction of the prolapsed urethra by some digital compression, and left the case to natural repair, with the hope that as the mucous membrane of the urethra was extremely fissured in many places, the healing of these fissures and the subsequent contraction of the cicatrices would yield sufficient support to the urethra to bring about a cure; and I may say that in that hope I have not been disappointed, since the child is now (seven months after the operation) quite well. Had I not found these fissures in the prolapsed mucous membrane, or sufficient loss of tissue to lead me to look for help from the healing process in effecting a cure, I should have cut off with scissors three or four strips of mucous membrane, radiating bladderwards from the orifice of the prolapsed urethra; and this is the line of treatment I should advise in similar but less severe cases of this kind. In the one I have related it was not, however, necessary.

In the subsequent progress of the case there is not much to record. For four days after the reduction of the prolapse the urine had to be drawn off by a catheter; at the end of that time the bladder resumed its power, but remained irritable, and was unable for about three weeks to retain its contents for more than two or three hours at a time. After that period everything progressed favorably. The soft parts about the urethra were swollen and painful for some days after the operation, but these symptoms soon disappeared, and the child, as I have reported, is now quite well.

I should like to add that, in the case related, the clitoris was unusually long and penile in form, with a well-made

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prepuce, suggesting that the condition of the urethra, which allowed such a rare affection as prolapse to take place, might have been due to some malformation of the passage. It would be well for future observers to note this point.

Remarks.—On looking up this subject, I find that similar cases to the one I have just related have been recorded, but they are clearly very rare, since amongst my numerous surgical friends in this country I have hardly found one who knows anything about them. Under these circumstances I have been induced to put my notes of the case together, with an epitome of the scanty literature of the subject.

M. Guersant is reported ('Bulletin général de thérapeutique,' October 15th, 1866, p. 307, and Ranking's 'Abstract' for 1867, p. 213) to have met with twelve or thirteen cases of prolapsed urethra in girls from two to twelve years of age. He attributes it to the following causes:

(1) Repeated straining, as in the cough of pertussis and chronic bronchitis, and in the repeated and forced attempts at defæcation consequent upon constipation.

(2) General debility.

Guersant has met with prolapse of the mucous membrane of the urethra in young girls during a long convalescence from acute disease and during chronic affections. The infants do not suffer much from this affection; there is sometimes a frequent desire to micturate, and scalding during the passage of urine. If the parts be examined, the vulva will be found redder than it usually is, and at the meatus urinarius will be observed a small rosy swelling, apparently proceeding from the interior of the canal, and the surface of which is formed of mucous membrane; at the centre of this little tumour there is an orifice through which a sound may be passed and carried into the bladder. This state of things may continue for a long time without producing any disturbance, but sometimes the swelling increases gradually, and gives off a sanguineous discharge which afterwards becomes puru-

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lent, its surface becomes irritated, and superficial sloughing may occur, with inflammation of the adjoining parts and vulvitis.

In the *treatment* of this affection Guersant prefers incision to cauterisation or the ligature, as the growth is by this means removed promptly and the child is sooner restored to its normal condition. The labia majora having been separated by an assistant, the prolapsed portion is drawn forwards either by a tenaculum or by a loop of thread, and then snipped off at one stroke of the scissors. If there be much hæmorrhage it may be arrested by the application of a solution of perchloride of iron, or by pressing for some seconds upon the part a small plug saturated with a styptic fluid. Frequent washing with cold water, and the occasional application of the stick of nitrate of silver, will suffice to produce cicatrisation.

Dr. G. E. Herman, of the London Hospital, published in the 'British Medical Journal' for February 9th, 1889, a case of prolapse of the mucous membrane of the female urethra which took place in a girl aged nine, who, from her early childhood, had had difficulty in holding her water any length of time, but had otherwise been well. On February 3rd, 1888, after the child had suffered for two or three days from a severe cough, which all domestic remedies failed to relieve, her mother was alarmed by finding a sanguineous discharge from the vulva. On February 6th, Mr. S. Welch, of Victoria Park Road, was sent for. He found a dark-red, cherry-like tumour in the situation of the meatus urinarius. On February 8th Dr. Herman saw the case with Mr. Welch. The tumour then consisted of a sort of thick frill, deep red in colour, projecting for rather more than half an inch from the lower part and sides of the meatus. At the upper part was an opening which the catheter entered. The tumour was not exceedingly tender, and the patient complained of no pain or difficulty in micturition. On February 14th, the state of things being much the same, except that there were some small sloughy spots on the most prominent part

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of the protrusion, the patient was anæsthetised, and with a wooden staff in the urethra, the prolapsed mucous membrane was cut off all round with the platinum knife of the Paquelin's cautery. The child recovered well, pain in micturition being much relieved by the child passing water in a warm bath.

Dr. Herman writes:—"The morbid condition in this case (prolapse or inversion of the urethral mucous membrane) is a rare one. The literature relating to it is scanty, and much of it unsatisfactory, because in reading many of the reported cases one cannot help thinking as to some that they were cases of vesical tumours projecting through the urethra; as to others, that they were simply urethral caruncles. Judging from the few cases that are well described, it seems to occur chiefly in children, and in the later years of childhood. It does not cause much trouble in micturition, but the protrusion is tender, although the tenderness is nothing like that of a urethral caruncle. The protruded mucous membrane often bleeds."

The method of *treatment* advised varies. Thus T. Emmet, of New York, recommends his "button-hole" operation; that is, making a button hole in the urethra from the vagina, pulling out the mucous membrane at this button hole (so as to pull back the protruding part) and cutting it off, then sewing up the "button-hole."

Dr. Herman observes upon this matter that "it seems to me more rational to cut off the protruded and congested mucous membrane, rather than some healthy mucous membrane higher up, to say nothing of its being a simpler and easier operation."

In 1890 (*vide* article in 'Brit. Med. Journ.,' vol i, 1890, p. 854) the subject was discussed at the Berlin Obstetrical Society, and Dr. Benicke described three cases.

The *first* was eleven years old, the prolapse was slight, and readily cured by the application of the actual cautery.

The *second* child was ten years of age and robust; the

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urethra was greatly dilated and the prolapse very marked; it was reduced, and the meatus made narrow by means of one suture.

The *third* patient was also ten years of age; the prolapse was extreme and irreducible. The protruding mucous membrane was cut away, and the edges of the wound were united by catgut sutures.

Frequent and severe hæmorrhage was the chief symptom in all these cases; dysuria and allied forms of suffering were absent.

The causation of the prolapse could not be traced.

Dr. C. Ruge and Professor Martin stated their belief that prolapse of the urethral mucous membrane was caused by the development of a vascular growth under the mucosa, which also underwent partial hypertrophy. Martin mentioned that cutting off the prolapsed tissue sometimes caused the formation of a troublesome cicatrix; Professor Veit had never seen this result.

Prolapse of the urethral mucous membrane, which is in no way related to caruncle, is certainly *rare before puberty*. Several distinguished authorities who joined in the discussion *had never seen the disease in children*.

Dr. T. Gaillard Thomas, in his 'Treatise on the Diseases of Women' published in 1891, p. 154, says that he has seen such cases in adults of enfeebled constitution and in little girls before the age of puberty. He gives also a drawing of such a case, but it is evidently not of a severe example. Beyond these extracts I can find no other records of this affection, and trust that what I have written may prove of interest and instruction.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. vi, p. 100.)

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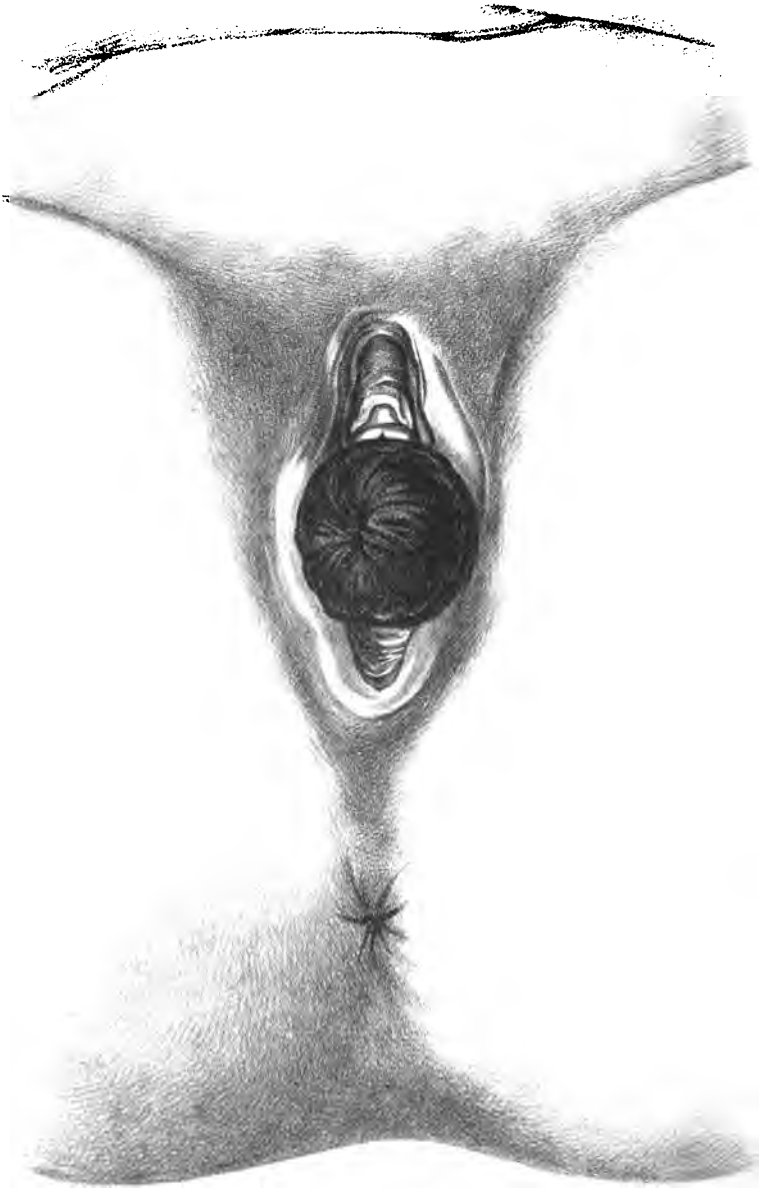

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UPON

TWO CASES OF URETERO-LITHOTOMY.

BY

EDWARD COTTERELL, F.R.C.S.,

SURGEON (OUT-PATIENTS) LONDON LOCK HOSPITAL, &c.

Received April 24th, 1894—Read May 8th, 1894.

In bringing these two cases of removal of calculus from the ureter before the notice of this Society, I would note that only a very few cases of the kind have been recorded, and that as operations of this nature are comparatively rare, all cases should be recorded in order that the proceeding may be put upon a more definite footing than it is at present.

The first case I have to record is that of Mrs. M—, sent to me by Mr. Wyllys, with symptoms of stone in the pelvis of the right kidney. The patient was sixty-one years of age, and was very stout. The kidney was explored on March 13th, 1893, when eighteen or twenty calculi were removed, the largest of which weighed over 200 grains. The kidney wound healed up in about ten days, and everything appeared quite satisfactory. She got up on April 15th, and that evening had a severe attack of renal colic on the right side. Very severe attacks of colic occurred at intervals for the next twelve days; and thinking, therefore, that there must be another

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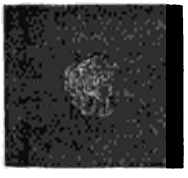
stone in the kidney which had escaped notice at the time of the previous nephro-lithotomy, I again explored the kidney on April 27th, but with a negative result. A probe was also passed about four inches down the right ureter, but did not hit any stone. In August her condition was as follows :—There was a renal fistula in the right loin, which had existed since the last operation. There was tenderness on pressure over the right inguinal region, but no acute attacks of pain. Vaginal and rectal examination failed to detect anything like a calculus impacted in the lower end of the ureter. Having determined that a calculus was impacted in the ureter between the points explored, on August 3rd I cut down upon it by an incision similar to the one used in the dead-house for tying the common iliac artery. The peritoneum was stripped up, and carried the ureter forwards with it. Owing to the obesity of the patient the wound was a very deep one, and it took a little searching to find the ureter. When this was recognised I traced it downwards towards the bladder, and, just beyond the level of the brim of the true pelvis, found the stone. With the finger and thumb I worked the calculus upwards until it was opposite the incision, and in a more manageable position, when I



extracted it through a small longitudinal incision. The wound was then closed in the ordinary manner, a drainage-tube being put in at the lower angle. The ureter was not sutured. No urine escaped by the wound, which quickly healed up. The calculus removed weighed four grains, and was very irregular in outline.

The second case is that of Mrs. W—, aged forty-four. She came under my care for internal piles, which I removed. When convalescent from this operation she told me that she had pain after passing water, and upon examining her bladder with a sound I detected what I took to be a foreign body coated with phosphates, as it did not give a clear clicking sound on being struck.

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Upon April 14th the patient was anæsthetised, but no stone or foreign body could be detected by the bladder sound. However, being sure that there was something in the bladder, although I failed to detect it with the sound, I dilated the urethra with Hegar's uterine dilators, and upon exploring the bladder with the finger two calculi could be felt impacted in the lower end of the right ureter. They did not project into the bladder. I removed them by cutting down upon the ureter through the roof of the vagina. No urine escaped through the wound, which quickly healed. The ureter was not sutured. The stones weighed fifteen grains, and from their appearance I should judge that they had been there some time. Indeed, I find that the patient had a severe attack of right renal colic last July, but did not pass a stone afterwards.

I have been able to discover ten other recorded cases of uretero-lithotomy, as given in the following table.

On glancing through the accompanying table, it will be seen that impaction occurred in three of the cases about two inches below the kidney: at the lower end of the ureter just before or whilst perforating the bladder wall in three cases, whilst in the remaining six the stoppage took place somewhere about the level of the brim of the true pelvis, where the ureter makes a turn forwards.

It may also be noted that all the cases recorded, where the impaction has taken place at the lower end of the ureter, have occurred in women, and up to the present no case has been noted of uretero-lithotomy in the male for stone impacted in this situation.

Recovery has occurred in all the cases except the two recorded by Thornton; in one of these death does not appear to have been due to the uretero-lithotomy, as the stone was extracted from the left ureter immediately after removing the right kidney for calculous pyo-nephrosis. In the other fatal case the urethra was dilated, and the

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Table of Cases of Uretero-lithotomy.

	Operator.	Sex.	Age.	Position of stone in ureter.	How removed.	Treatment of ureter.	Result.	Where recorded.
1	Kirkham	M.	58	½ inch above where it crosses external iliac vessels	Extension of nephrotomy wound	Not sutured	Recovery	Lancet, March 16th, 1889.
2	Twynam	M.	8	2 inches from bladder	Incision similar to ligature of common iliac	Sutured	„	Brit. Med. Journ., Feb. 1st, 1890.
3	Godlee	F.	26	2 inches below kidney	Nephrotomy incision	„	„	Trans. Clin. Soc., vol. xxii.
4	Arbuthnot Lane	F.	—	Where it crosses external iliac vessels	Laugenbuch's incision	„	„	Lancet, 1890, Nov. 8th.
5	Bardenheuer	—	—	Middle third	Flank incision	„	„	Centralblatt fur Chirurg., March, 1882.
6	Thornton	F.	—	Lower end	Dilatation of urethra; removal through bladder	—	Ureteral fistula; death in 14 days, from exhaustion	Harveian Oration.
7	„	F.	—	Halfway down	Found during removal of right kidney by abdominal operation; removed by small lumbar incision	Not sutured	Death 6th day; broncho-pneumonia	Ditto.
8	Cabot	M.	40	2 inches below kidney	Nephrotomy incision	„	Recovery	Boston Med. and Surg. Journ., 1890, p. 247.
9	„	F.	39	Lower end	Through roof of vagina	„	„	Ditto, p. 613.
10	Hall	F.	—	2½ inches below kidney	Diagnosed by abdominal section; removed by lumbar incision	„	„	Trans. Amer. Assoc. Obst. and Gyn., 1890.
11	Cotterell	F.	61	Little below where it crosses external iliac vessels	Incision similar to ligature of common iliac	„	„	This paper.
12	„	F.	44	Lower end	Through roof of vagina	„	„	This paper.

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stone cut out through the bladder, but a ureteral fistula resulted, the urine discharging into the pouch of Douglas. The woman went out of her mind, and died of exhaustion a fortnight later.

Impacted stone in the ureter gives rise to symptoms very like those of stone in the kidney, and in all the recorded cases where the stone was impacted somewhere in the upper three fourths of that tube the kidney has been explored first, with the expectation of finding a calculus there, and it has been owing to the negative evidence resulting from this measure that exploration of the ureter has been undertaken. Pain is a prominent symptom, renal colic being well pronounced. When the stone becomes impacted at any point the pain is generally most severely felt at this particular part. There may be frequency of micturition, or the very reverse condition may occur from suppression of urine; and in this case, if the impacted calculus is not removed, uræmic symptoms will supervene.

The lower portion of the ureter can be felt and explored in the female by the vagina, and in the male by the rectum, but in the latter only the last half or three quarters of an inch can be so felt.

Given a large stone and a thin person it might be possible perhaps to feel the stone through the abdominal walls, but this is very doubtful, though the site of the impaction may be sometimes more or less localised by the patient's sensations during abdominal palpation; for a constant tender spot at one point, with pricking pain when that spot is manipulated, will generally determine the whereabouts of the stone. In two cases (Hall and Lane) the diagnosis has been made and the situation of the stone determined by abdominal section.

The question now arises, what is the best way to remove a calculus which is impacted in the ureter? In the case of stones within two to three inches from the kidney, they simulate so closely stone in that organ that a nephrotomy will be generally performed, and failing to

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find a calculus there, the upper part of the ureter can be explored through this wound.

When the stone is impacted lower down—that is to say, from three inches below the kidney to about two inches from the lower end, the ureter can be explored by an incision similar to that by which one is taught to tie the common iliac artery. It is important to bear in mind the relation of the ureter to the peritoneum, for the former is adherent to and always strips up with the latter, when this is separated from the parts behind. If the right side is being operated upon, the proximity of the vena cava should be remembered.

The last two or three inches of the ureter can be reached in the female by an incision through the roof of the vagina without opening the peritoneal cavity, and this is preferable to extracting the stone through the bladder. If a stone is impacted in this region in the male, I would suggest that it can be reached by an incision similar to that proposed by Zuckerkandl for the removal of the seminal vesicles. This consists of a semicircular incision in the perinæum, separation of the anterior surface of the rectum, incision of the anterior fibres of the levator ani so as to lay bare the prostate, when, by means of a sound in the bladder, the base of that viscus can be made prominent in the wound, and a little dissection on one side will expose the lower end of the corresponding ureter.

There does not seem to be any object in stitching up the wound in the ureter, but care should be taken to make the incision into it as small as possible. Wounds of the ureter heal very readily, and the suture might become the nucleus for a future calculus.

[*Note.*—Since the foregoing paper was written, Mr. Wheelton Hind has recorded ('British Medical Journal,' vol. i, p. 960) a case of total suppression of urine due to a calculus impacted in the upper end of the right ureter; the left kidney being atrophied, with the pelvis enclosing a calculous deposit weighing 360 grains. The impacted

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calculus was removed by an incision down to the kidney in the right loin, and then cutting on to the stone in the upper part of the ureter. A nephro-lithotomy was afterwards performed as there was a calculus in the pelvis. The ureter wound was not sutured. The patient died fifty-eight hours after the operation.]

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. vi, p. 103.)

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THE INFLUENCE OF DIFFERENT KINDS OF SOIL

ON THE

COMMA AND TYPHOID ORGANISMS.

BY

R. DEMPSTER, M.B.,

COBB RESEARCH SCHOLAR, 1893.

(*From Professor Macfadyen's Laboratory, British Institute of Preventive Medicine.*)

(COMMUNICATED BY DR. MITCHELL BRUCE.)

Received May 8th—Read May 22nd, 1894.

THE influence of external conditions upon pathogenic organisms is a subject that has naturally engaged the attention of bacteriologists for a considerable time; and the important bearing which researches in this direction have upon the epidemiology of disease will prove a sufficient stimulus for prolonged investigation in the future. Of such enquiries we cannot have too many, as upon the facts which they succeed in demonstrating the foundations of hygiene will ultimately rest.

It was the importance more than the novelty of the subject that led me to undertake this investigation, the results of which I now have the honour to lay before you. The work was undertaken with a view to endeavour to answer the following special question:

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Has the soil in itself any action favourable or injurious to the life of the comma bacillus of cholera Asiatica, and the bacillus of typhoid fever? or does the length of life of these organisms in the soil simply depend upon the amount of moisture that may be present?

The action of the saprophytic bacteria present in the soil was left out of consideration altogether. For the purposes of this research *sterilised soils* alone were used.

The experiments were carried out with four different kinds of soil, namely, white crystal sand, yellow sand, garden earth, and peat.

First Series of Experiments.

To find the length of time that the soils should be exposed to steam in Koch's apparatus to render them sterile.

Samples of the soils to be used in this research were placed in glass vessels provided with cotton plugs, and sterilised. After sterilisation gelatine-plate cultures, also beef-broth inoculations, were made.

It was found that a small quantity of soil can be sterilised by steam in Koch's apparatus, if exposed for one hour on three successive days to the temperature of boiling water.

Second Series of Experiments.

To find how long comma bacilli will live in a natural dry soil.

Three kinds of soil, namely, white crystal sand, yellow sand, and garden earth were passed through a sieve to remove stones, &c.

A measured quantity of each kind of soil was put into sterilised test-tubes and sterilised.

Mode of procedure.—The cholera bacilli were suspended in ¾ per cent. salt solution, or in distilled water, and for this purpose agar-agar cultures were

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used, and the growth mixed with the sterilised salt solution or distilled water so as to form a faint milky emulsion; a measured quantity of this emulsion was added to each of the soils, not exceeding 1 c.c.

FIG. 1.

The tubes were either kept at room temperature or at 22° C. From time to time gelatine-plate cultures were made from the soils and, after incubation, examined for the presence of bacilli.

The *peptone water medium* as used by Koch was found of great service in the examination of the soil for comma bacilli. This peptone water was made as follows:— 1 per cent. peptone, and 1 per cent. sodic chloride, were dissolved in 100 parts of water and the fluid rendered strongly alkaline.

The comma bacilli are more readily detected by this medium than by the ordinary plate methods. I found in the course of my experiments that the comma bacilli were often detected by means of peptone water cultivations when gelatine plates made from the same soil gave negative results. The peptone water, after being inoculated with the soil, was kept at blood heat. The comma bacilli multiply very rapidly in this medium, and rise to the surface: the result is that if one examines the peptone tubes after eight to twelve hours, one finds the comma bacilli in large numbers in the upper portion of the fluid; they are then readily detected by the microscope, and if plates be made from this peptone water there is no difficulty in obtaining typical colonies of the comma bacillus. The following method gives the best results, namely, first, to add the soil to peptone water, and then after twelve hours to make plates from the peptone water, instead of making plates directly from the soil.

By means of the methods described above, the results to be detailed were obtained. This series of experiments

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was made with tubes plugged with cotton-wool (Fig. 1) and kept at 22° C. Consequently the soils lost their moisture.

In the white crystal sand, the yellow sand, and the garden earth, the comma bacilli were alive on the third day.

In the above three soils the comma bacilli were dead on the fifth day. Therefore the comma bacilli lived in these soils from three to five days.

The soils in the above experiments were air-dry, and only contained traces of moisture when the cholera emulsion was added to them.

Third Series of Experiments.

To find how long comma bacilli will live in a soil that has been rendered moist, without, however, containing any excess of moisture.

A measured quantity of white crystal sand, yellow sand, and garden earth was placed in sterilised test-tubes; these were made moist, and sterilised. Control experiments were then made to make sure that the soils were sterile before being used. To each of the three kinds of soil was added 1 c.c. of the watery emulsion of comma bacilli.

These experiments were made with tubes plugged with cotton wool and kept at 22° C.; consequently the soils gradually lost the moisture they contained.

The results obtained with gelatine plates were as follows:

In moist white crystal sand comma bacilli were found up to the seventh day. In moist yellow sand and in moist garden earth the comma bacilli were alive on the thirty-third day.

From these experiments it will be seen that the comma bacilli *lived longer in the moist soils* than was the case in the air-dry soils.

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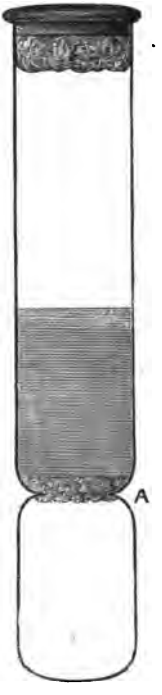
Fourth Series of Experiments.

To find the length of time comma bacilli will live in a soil when any excess of moisture in the soil is allowed to escape through the soil, but where loss of moisture does not take place from the surface of the soil.

Instead of using ordinary test-tubes a special form of tube was obtained, as shown in Fig. 2. At the constriction A a layer of cotton-wool was placed, to form a support for the soil above it and also to allow any excess of moisture in the soil to pass into the lower part of the tube. The tubes were plugged with cotton-wool and sterilised.

Fig. 2.



In three such sterilised tubes a measured quantity of soil was placed, and well sterilised.

The soils used were white crystal sand, yellow sand, and garden earth.

About 3 c.c. of an emulsion of comma bacilli was added to each of the soils; the tubes were provided with tight, well-fitting india-rubber caps to prevent loss of moisture, and kept at a temperature of 18° to 20° C. Any excess of moisture was able to pass through the layer of cotton-wool under the soil into the lower part of the tube.

Gelatine-plate cultures and peptone water inoculations of the soils were made from time to time to see if the bacilli were alive.

The results were as follows:

In white crystal sand the comma bacilli were found alive on the twenty-eighth day.

In yellow sand and in garden earth the comma bacilli were found alive on the sixty-eighth day.

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Fifth Series of Experiments.

To find how long the comma bacilli will live in dried soils.

White crystal sand, yellow sand, garden earth, and peat having been passed through a fine sieve, were then *dried in porcelain dishes over the Bunsen flame*, and a measured quantity of each was placed in sterilised test-tubes and the whole sterilised. To each of the four kinds of soil was added 1 c.c. of an emulsion of comma bacilli. These experiments were made with tubes plugged with cotton-wool and kept at blood heat, and under these conditions the soils quickly lost any added moisture.

The results obtained with gelatine-plate cultures were as follows:

In none of these dried soils were comma bacilli found at the end of twenty-four hours.

In one or two experiments they were found alive in the white crystal sand, yellow sand, and garden earth *after twenty-four hours*, but *not after forty-eight hours*.

The comma bacilli did not therefore live in the dried soils longer than one to two days. *The only exception was peat, in which the comma bacilli were invariably dead within twenty-four hours.* The results obtained with a peat soil will be specially referred to later on.

If we compare the above series of experiments we get the following results:

On a dried soil, the comma bacilli lived one day.

On an air-dry soil, the comma bacilli lived three to five days.

On a moist soil in which a gradual loss of moisture took place, the bacilli lived seven to thirty-three days.

On a moist soil in which little or no loss of moisture took place, the bacilli lived twenty-eight to sixty-eight days.

On a moist soil, in which, however, loss of moisture was taking place gradually, the bacilli were not alive in a

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control experiment made on the fifty-eighth day; whilst on a moist soil losing little or no moisture, it will be seen that the bacilli were alive on the sixty-eighth day.

These experiments point to the close connection existing between the amount of moisture present in the soil and the length of life of the bacilli.

With a view of controlling these results a series of parallel experiments were made with white crystal sand. The results obtained were as follows:

In the white crystal sand *which had lost its moisture*, the comma bacilli were alive on the third day, but were dead on the eighth day.

In the white crystal sand *which was kept moist*, the comma bacilli were still alive on the forty-seventh day.

Therefore the comma bacilli lived longer in the soil that was kept moist.

A series of experiments was made to find the amount of moisture that is present in each of the soils used in these experiments.

It was found that—

The white crystal sand contained	0·1	per cent.	of	moisture.
The yellow sand	,, 1·18	,,		,,
The garden earth	,, 2·2	,,		,,
The peat	,, 25·34	,,		,,

Each of these soils before being weighed was passed through a fine sieve to remove stones, &c., and it is in this fine condition that the soils have been used in these experiments.

Sixth Series of Experiments.

To find the rate at which the soils lose their moisture by evaporation after the addition of an emulsion of comma bacilli, and kept at 18° to 20° C.

White crystal sand, yellow sand, garden earth, and peat were used in these experiments.

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The results were as follows :

		per cent.		
White crystal sand	lost	0·304	of its moisture	per day.
Yellow sand	,,	0·292	,,	,,
Garden earth	,,	0·302	,,	,,
Peat	,,	0·297	,,	,,

These experiments show that when these four soils are placed under the same conditions of temperature they lose their moisture at a certain rate, *which is about the same in all the soils.* But in nature, owing to local and seasonal conditions, the rate of loss would not be the same from all soils. Thus good drainage will carry off the moisture more quickly; and one soil may drain more quickly and more thoroughly than another, thus parting with its moisture more quickly.

The amount of natural moisture in each of these four kinds of soil varied greatly, and that soil which contains the least moisture (or which can part with its moisture in the shortest time by drainage or evaporation) is the one in which the bacilli might be expected to live the shortest time.

Under equal conditions of temperature, the *loss* of moisture by evaporation was about the same in white crystal sand, yellow sand, garden earth, and peat. The *amount* of natural moisture present in these soils, however, was different. The white crystal sand contained the least, garden earth and peat the most moisture. Sand being a surface soil will lose its moisture most quickly by evaporation and drainage, and in it under natural conditions the life of the organisms is therefore likely to be the shortest.

Seventh Series of Experiments.

To find the relation between the amount of moisture in the soil and the life of the comma bacillus.

Rationale of the process.—Having obtained the weight of a sterilised test-tube with its cotton-wool plug, a given

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quantity of soil was weighed out and placed in this tube, and then sterilised; the emulsion of cholera bacilli was added, and the tube weighed so as to obtain the weight of the cholera emulsion added. The soil was kept at a temperature of 18° to 20° C., and weighed daily; the loss of weight represented the loss of moisture. Gelatine-plate or peptone-water cultures of the soil were made from time to time, the weight being also noted, until the bacilli were found to be dead. We thus obtained the time when the bacilli were last found alive, and also the amount of moisture that had escaped from the soil, and consequently the amount of moisture left in the soil and the point of dryness of the soil below which the bacilli cease to live.

This series of experiments was performed with white crystal sand and yellow sand, in tubes plugged with cotton wool and kept at 18° to 20° C. so that the soils gradually lost their moisture; as well as in tubes closed with a tight, well-fitting india-rubber cap (and kept at 18° to 20° C.), by means of which the loss of moisture was very considerably delayed.

The results obtained with white crystal sand were as follows:

In the white crystal sand *where the loss of moisture went on quickly* comma bacilli were alive on the twenty-seventh day, but were dead on the thirtieth day: and it was found that on the twenty-seventh day, when the bacilli were alive, the sand contained 1·572 per cent. of moisture; while on the thirtieth day, when the bacilli were dead, the soil only contained 0·66 per cent. of moisture.

In the white crystal sand *where the loss of moisture was almost prevented* comma bacilli lived much longer, and were alive on the 174th day, and the sand contained 7·1 per cent. of moisture.

The results obtained with yellow sand were as follows:

In the yellow sand *where the loss of moisture was rapid* the comma bacilli were dead on the twelfth day, when the soil contained 1·44 per cent. of moisture. And in the yellow sand *where the loss of moisture was almost pre-*

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vented the bacilli were alive on the thirty-sixth day, when the soil contained 8·659 per cent. of moisture.

These experiments illustrate the close relation existing between the amount of moisture present in the soil and the length of life of the micro-organisms. For example, in white crystal sand containing 7·1 per cent. of moisture the bacilli were found alive up to the 174th day; in white crystal sand containing 1·57 per cent. of moisture the bacilli were found alive up to the twenty-seventh day, but on the thirtieth day, when the amount of moisture had fallen to ·66 per cent., the bacilli were dead.

Eighth Series of Experiments.

To find the length of life of the comma bacilli in sterilised ·75 per cent. salt solution.

The comma bacilli were obtained from pure cultures on nutrient agar-agar.

10 c.c. of ·75 per cent. salt solution was placed in a sterilised test-tube and sterilised.

Into this sterilised salt solution was introduced some of the pure culture of comma bacilli, and kept at 18° C.

When the comma bacilli had been in this salt solution for 159 days, a tube of peptone water was inoculated from the salt solution and placed in the 37° C. incubator for twenty-four hours. On examination the peptone water was cloudy, and had a distinct surface pellicle. A microscopical preparation of this pellicle revealed comma bacilli.

The comma bacilli had remained alive in the salt solution for 159 days; beyond which time the experiments were not carried.

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Ninth Series of Experiments.

To find the length of life of the comma bacilli in sterilised acid urine.

Into sterilised test-tubes about 20 c.c. of fresh acid urine was placed, and sterilised in the usual way. The tubes were then inoculated with a pure culture of the comma bacilli. Some of these tubes were kept at blood heat (37° C.); others at 22° C.

Gelatine-plate cultivations and peptone-water inoculations were made from time to time with the urines, and examined microscopically.

The results obtained were as follows:

The comma bacilli lived in urine at blood heat for 14 days.
" " " " 22° C. " 29 "

TYPHOID.

Tenth Series of Experiments, with the Bacillus of Typhoid Fever.

To find how long typhoid bacilli will live in a natural dry soil.

Three kinds of soil, namely, white crystal sand, yellow sand, and garden earth were passed through a sieve to remove stones, &c.

Into sterilised Erlenmeyer flasks and test-tubes a measured quantity of each kind of soil was put, and sterilised.

Inoculations from control flasks proved that the soil contained no living organisms.

An agar-agar culture of typhoid bacillus was taken and a faint milky emulsion made in sterilised ·75 per cent. salt solution.

To each of the soils was added 1 c.c. of an emulsion of typhoid bacilli.

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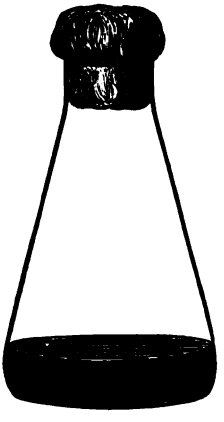
These experiments were made with flasks and tubes plugged with cotton wool (Fig. 3) and kept at 22° C.; consequently the soils gradually lost their moisture.

Fig. 3.



The results obtained with gelatine-plate cultures were as follows:

In the white crystal sand the typhoid bacilli died between the ninth and the eleventh days.

In the yellow sand they died between the eighteenth and twentieth days.

In the garden earth they died between the fourteenth and the eighteenth days.

Tenth Series of Experiments (*continued*).

To find the length of time typhoid bacilli will live in peat.

The peat having been passed through a sieve, a measured quantity was placed in sterilised test-tubes, and the whole sterilised. To each was added 5 c.c. of an emulsion of typhoid bacilli. The tubes were plugged with cotton wool and kept at 32° C.

The results obtained with gelatine-plate cultures were as follows:

The typhoid bacilli were dead at the end of twenty-four hours.

Typhoid bacilli were found alive at the end of one hour, but not at the end of twenty-three hours.

In the case of the comma bacilli it was found that they died on a peat soil *within twenty-four hours*.

The typhoid organisms were also dead within twenty-four hours. These results were quite independent of the amount of moisture present in the peat. The peat had therefore a marked destructive action on the micro-organisms even when an abundance of moisture was

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present. Peat was *the only one* of the soils experimented with which proved distinctly inimical to the life of these microbes, and its action was rapid and energetic.

Eleventh Series of Experiments.

To find the length of time that typhoid bacilli will live in a soil that has been rendered moist.

Into sterilised test-tubes was put a measured quantity of white crystal sand, yellow sand, and garden earth; these were rendered moist, and then sterilised.

To each of the three kinds of soil 1 c.c. of an emulsion of typhoid bacilli was added.

The experiments were made with tubes plugged with cotton wool, and kept at 22° C.; and the soils gradually lost their moisture.

The results obtained with gelatine plates were as follows:

In the moist white crystal sand typhoid bacilli were alive on the twenty-third day, but were dead on the twenty-seventh day.

In the moist yellow sand and moist garden earth the typhoid bacilli were alive on the forty-second day.

Twelfth Series of Experiments.

To find the length of time typhoid bacilli will live in dried soils.

White crystal sand, yellow sand, and garden earth having been passed through a fine sieve and well dried, a measured quantity of each was put into sterilised test-tubes, and the whole sterilised.

To each of the three kinds of soil was added 1 c.c. of an emulsion of typhoid bacilli.

These experiments were made with tubes plugged with cotton wool and kept at blood-heat (37° C.); and the added moisture gradually diminished.

The results obtained with gelatine plates were as follows:

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The typhoid bacilli were found to be alive in all three kinds of soil on the seventh day, but were dead in all three on the ninth day.

To sum up—On a *dry* soil where evaporation was allowed to take place the following results were obtained:

In white crystal sand the bacilli were found up to the ninth day, in yellow sand up to the eighteenth day, and in garden earth up to the fourteenth day.

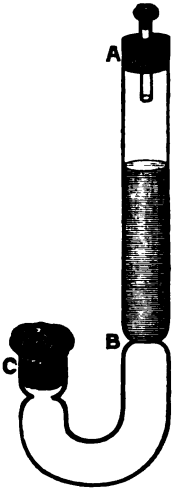
On the *moist* soils, on the other hand, the following were the results:

In moist white crystal sand the typhoid bacilli were alive on the twenty-third day; in yellow sand and on garden earth on the forty-second day.

On soils which had been deprived of their moisture the bacilli were only found up to the seventh day.

Thirteenth Series of Experiments, made to test the Filtering Capacities of the Soils for Bacteria.

Fig. 4.



Glass tubes were used, as shown in Fig. 4. At the constriction B was placed a layer of wire gauze to support the soil, and on this rested a layer of soil six inches in depth. The end of the tube C was plugged with cotton-wool. The end A was provided with a perforated rubber stopper; through the perforation was passed a glass tube having its external end plugged with cotton-wool.

A layer of six inches of the soil to be tested was introduced. The soils used were white crystal sand, yellow sand, and garden earth. The open ends of the tubes were plugged, and the tubes of soil well sterilised by steam.

An emulsion of typhoid bacilli was made by taking

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an agar-agar culture of the bacilli, and mixing the growth with ·75 per cent. salt solution till a faint milky emulsion was produced.

This emulsion of typhoid bacilli was taken up in sterilised pipettes and placed on the surface of the soils. Gelatine-plate cultures were made of the fluid that passed through the soils (the filtrates), and these plates were examined for typhoid bacilli, or for counting the colonies.

1st. 4 c.c. of this typhoid emulsion was placed on the surface of the *white crystal sand.* Some of it passed through the soil, and a gelatine-plate culture was made of the filtrate, and colonies of typhoid bacilli developed in the plate. The typhoid bacilli were therefore carried through six inches of the white crystal sand.

2nd. 2 c.c. of an emulsion of typhoid bacilli was poured on the surface of the *yellow sand,* and four days later some sterilised distilled water was allowed to fall on the sand to see if it would carry down any typhoid bacilli through the soil; some of this water passed through the six inches of yellow sand, and a gelatine-plate culture was made of this filtrate; on incubation this plate develope typical colonies of the typhoid bacilli.

3rd. 2 c.c. of the emulsion of typhoid bacilli was placed on the *garden earth,* and four days later some sterilised distilled water was allowed to fall on the surface of the soil, to see if it would carry down any typhoid bacilli; some of this water passed through the soil, and a gelatine-plate culture was made of this filtrate on which developed colonies of the typhoid bacilli. The typhoid bacilli were thus carried through six inches of garden soil.

Resumé.—Typhoid bacilli passed through six inches of white crystal sand, and were found in the filtrate on the fifth day.

Typhoid bacilli passed through six inches of yellow sand, and six inches of garden earth, and were found in the filtrates on the fourth day.

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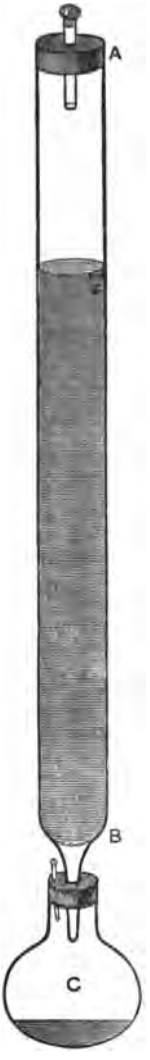
FIG. 5.



FOURTEENTH SERIES OF EXPERIMENTS.

Can the bacilli be carried by a current of water through a layer of sand two feet six inches in depth?

The sand on the surface of a filter-bed is 2 feet 6 inches in depth.

A glass tube was used, as shown in Fig. 5. At the constriction B was placed a layer of wire gauze to support the soil, and on this gauze rested a layer of white crystal sand 2 feet 6 inches in depth. The end of the tube at A was provided with a perforated rubber stopper, through which passed a glass tube having its external end plugged with cotton wool. The lower end of the tube was pointed, and passed through a rubber stopper into a glass flask; this rubber stopper should have a perforation through it containing a small glass tube plugged with cotton wool, so as to allow air to escape from the flask while its place is being taken up by water falling into the flask from the filtration tube.

The white crystal sand for this experiment was sterilised by heating it in a vessel over the Bunsen flame.

The filtration tube was sterilised with corrosive sublimate solution, and then washed well with sterilised distilled water.

The flask was freed from micro-organisms by dry heat in the sterilising oven.

White crystal sand, to the depth of 2 feet 6 inches, was placed in the filtering tube. This

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sand was quite dry, all moisture having being driven off while sterilising it over the Bunsen flame, so that to resemble a working filter it was made moist again with water. A known quantity of sterilised distilled water was poured on the surface of the sand till it began to fall into the flask; the tube was left for twenty-four hours, so that all excess of water might drain from the soil; the flask was then removed, and a freshly sterilised one took its place. The next step was to see if water containing comma bacilli would pass through this 2 feet 6 inches of white crystal sand; for this purpose an emulsion of comma bacilli was made with 250 c.c. of sterilised distilled water. The whole of this emulsion was by degrees poured on the surface of the sand, the time taken being one hour and a quarter for the emulsion to pass through the sand into the flask below. Gelatine-plate cultures and peptone-water inoculations were made of the filtrate, and comma bacilli were found.

The comma bacilli were therefore carried by the water through 2 feet 6 inches of white crystal sand. It was found that the dry sand retained 60 c.c. of the sterilised distilled water that was poured on it to make it moist, and that after pouring 250 c.c. of the emulsion of cholera bacilli on to the sand 250 c.c. of fluid passed into the flask, so that at the end of this experiment the sand still retained 60 c.c. of moisture.

A gelatine-plate culture was made of the cholera emulsion before it was poured on to the sand; $\frac{8}{100}$ c.c. of the emulsion was used to make this plate. A gelatine-plate culture was also made of the fluid that had passed through the sand, $\frac{6}{100}$ c.c. of the filtrate being used for this purpose. These two plates were made the same day, and on examination the plate made from the filtrate did not contain nearly so many colonies of the comma bacilli as the plate made from the emulsion, although twice the quantity of filtrate was used to make the plate. Therefore, although some comma bacilli passed

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through 2 feet 6 inches of sand, many have remained in the soil.

The next point was to determine if the comma bacilli remaining in the sand would be carried down into the flask by pouring some water on to the surface of the sand.

Twenty-four hours after the emulsion of comma bacilli had been placed on the soil, 250 c.c. of sterilised distilled water was poured by degrees on to the surface of the sand; it passed through the sand in fifteen minutes into a freshly-sterilised flask; this may be called the "second filtrate," and measured 250 c.c. A peptone-water inoculation on examination showed the presence of comma bacilli.

Although the sand therefore held back many comma bacilli during the first filtration of water contaminated with the bacilli, a further supply of fresh water on the surface of the sand carried down some of those bacilli that had been held back, and the second filtrate also contained comma bacilli.

Four days after the emulsion of comma bacilli had been placed on the soil, another 250 c.c. of sterilised distilled water was poured on the surface of the sand, to see if any more comma bacilli would be carried down into another freshly sterilised flask. The water was poured on the sand quickly, and at the end of nine minutes all the water had entered the surface of the sand, and was beginning to fall into the flask. This is the "third filtrate," and measured 250 c.c. The water fell from the soil into the flask in drops, not in a stream. A peptone-water inoculation on examination showed the presence of comma bacilli.

From the above it is seen that even a second supply of water on the surface of the filter carried down more comma bacilli, and that the third filtrate contained bacilli.

On the eighth day after the emulsion of comma bacilli had been placed on the soil, another 250 c.c. of sterilised distilled water was poured on the surface of the sand, to see if any more comma bacilli would be carried down from the soil. The fluid passed through the sand, and this

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fourth filtrate measured 245 c.c. A peptone-water inoculation on examination showed that there were *no* comma bacilli in this fourth filtrate.

The filtering capacity of filter beds depends upon the upper layer of sand. Fresh sand is not an efficient filter for micro-organisms; it is only when the suspended matter present in the water sinks and forms a film on the surface of the sand, that the bacteria present in the water are held back to any extent. The bacteria also establish themselves in the pores of the sand, and in this way also render the sand more impervious to the passage of bacteria. It will be seen, in the above laboratory experiment that, using the same sand filter two and a half feet thick, the first, second and third filtrates contained comma bacilli, but the fourth filtrate contained none.

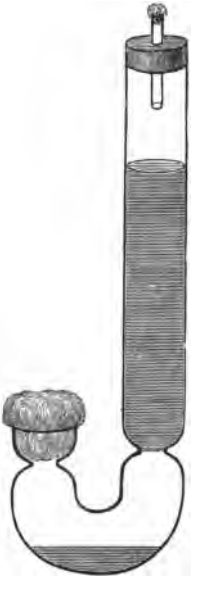
Fifteenth Series of Experiments.

To find the number of bacilli that are held back during filtration of cholera-contaminated water through six inches of white crystal sand, the sand being dry as it would be in a new filter.

White crystal sand was placed in a filtration tube to the depth of six inches; the tube was plugged (Fig. 6), and the whole well sterilised by steam.

With a sterilised pipette, 20 c.c. of an emulsion of comma bacilli was placed on the surface of the sand; at the end of two minutes some of the emulsion had passed through the sand, and at the end of five minutes filtration appeared to be completed; the amount of filtrate measured 10 c.c. It should be noted that this sand is in

Fig. 6.



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its natural condition; no water had been poured on it, so its pores were not saturated with moisture. It resembled a newly-made filter, or a surface of sand after dry weather.

The result with a filter of dry sand six inches thick was that 99·6 per cent. of the comma bacilli were held back.

Sixteenth Series of Experiments.

To find the number of bacilli that are retained in a filter of white crystal sand six inches in thickness when the sand is in a moist condition.

White crystal sand was placed in a filtration tube to the depth of six inches, the plugs were then placed in the two ends of the tube, and the whole sterilised in Koch's steam apparatus.

Sterilised water was poured on the sand till the whole was moistened, and the water passed through the sand in the bend of the tube. The soil was allowed to drain till the next day, when this water was decanted from the bend of the tube, and the bend was sterilised by heating it in the Bunsen flame in case air-borne germs had entered it during decanting.

With a sterilised pipette 13½ c.c. of an emulsion of comma bacilli was placed on the sand; in one minute and fifteen seconds some of the emulsion or filtrate had passed into the bend of the tube. The amount of filtrate measured exactly 13½ c.c.

A gelatine-plate culture was made of some of the cholera emulsion before it was poured on the sand, to ascertain the number of bacteria it contained before being filtered; $\frac{8}{100}$ths of a c.c. of the emulsion was used to make the plate. At the same time a gelatine-plate culture was also made of some of the filtrate, to ascertain the number of colonies that would be produced after filtration; $\frac{8}{100}$ths of a c.c. of the filtrate was used to make this plate. Colonies of comma bacilli developed in these plates, and they were counted as before.

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The plate made with $\frac{8}{100}$ths c.c. of the emulsion contained 1425 colonies, so that 1 c.c. of it contained 47,500 micro-organisms before being filtered.

The plate made with $\frac{8}{100}$ths c.c. of the filtrate contained only 48 colonies, so that 1 c.c. of it contained 1600 bacteria after filtration.

As each c.c. of the cholera emulsion is filtered through six inches of sand, 45,900 comma bacilli (47,500 less 1600) are held back by the sand; that is to say, 96·6 per cent. of the comma bacilli are held back by a filter of moist white crystal sand six inches in thickness.

The results of these experiments with a moist sand filter, then, showed that 96·6 per cent. of the comma bacilli were held back.

Seventeenth Series of Experiments.

To determine the number of bacilli that are retained by a filter of yellow sand six inches in thickness when the sand is in a moist condition.

It was found that the moist yellow sand held back 99·9 per cent. of the comma bacilli, as compared with 96·6 per cent. held back by the moist white crystal sand.

Eighteenth Series of Experiments.

To determine the number of bacilli that are retained in a filter of garden earth six inches thick when the soil is in a moist condition.

It was found that moist garden earth held back 89 per cent. of the comma bacilli.

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Nineteenth Series of Experiments.

To find the number of bacilli that are retained by a filter of peat six inches in thickness.

The result was that no comma bacilli were found to pass through a filter of moist peat six inches in thickness, or I would rather say no *living* bacilli passed through the peat filter. In my previous experiments I found that the peat quickly killed the comma and the typhoid organisms.

The following are the results of the filtering experiments:

A filter, six inches thick, composed of—

		Per cent.	
White crystal sand (dry)	holds back	99·6	of the comma bacilli.
,, ,, (moist)	,,	96·6	,,
Yellow sand	,,	99·9	,,
Garden earth	,,	89	,,
Peat	,,	100	,,

Results.

I have now given the essential points in my investigation, and it only remains to sum up shortly its results. I found that white crystal sand, yellow sand, and garden earth have *no* marked favourable or injurious action on the life of the comma bacillus of cholera Asiatica, nor on the bacillus of typhoid fever. The all-important factor determining their length of life in these soils is *moisture*. We saw how the comma bacilli quickly died when planted on a given *dried* soil, and how their life on the same soil was prolonged by simply adding water to the soil in question. These results apply also to the typhoid organisms. *Peat*, on the contrary, proved very deadly to both the comma bacilli and the typhoid bacilli. Peat was the only one of the four soils which, *per se*, and independently of the amount of moisture present, exercised a distinctly inimical action on the comma and typhoid bacilli.

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In sterilised salt solution the comma bacilli were alive on the 174th day. This experiment I was not able to carry further; but it seems proved by Koch and others that in fluid media at any rate the presence of sodic chloride greatly favours the life of the comma bacilli.

As regards urine, the bacilli lived longer at 22° C. than at blood heat.

All these experiments were carried out with *sterilised* soils.

The influence of the *saprophytic bacteria in the soil* on the life of pathogenic organisms is now being investigated in Professor Macfadyen's laboratory, and the results will form a sequel to those which I have detailed.

The filtering experiments showed that when the soils were used in a *pure* state moist yellow sand proved the most efficient filter for the micro-organisms, whilst peat not only retained but killed the bacilli.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. vi, p. 141.)

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A REVIEW OF CASES MANIFESTING PAIN AT THE HEART,

OR

MORBID ACCELERATION OF THE HEART'S CONTRACTION (TACHYCARDIA),

SUBSEQUENTLY TO INFLUENZA.

BY

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In this communication I propose to submit to review and analysis a series of cases, all of which have been under my own observation, wherein pains in close relation with the heart or disturbances of the cardiac rhythm in the sense of morbid acceleration have been manifested subsequently to attacks of influenza. In some of the cases the symptoms occurred in immediate sequence to the attack, in others more or less considerable periods elapsed before they were manifested, but in all the chain of evidence appeared to indicate the relation of cause and effect.

Reviewing 100 cases observed by myself, in which affections of the heart were initiated by influenza, I found them thus distributed:—Pain referred to the heart, 23 cases; morbid acceleration of the heart's contractions (tachycardia), 37 cases; pronounced irregularity of the

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heart's action (arhythmia), 25 cases; abnormal retardation of the rate of pulsation (bradycardia), 5 cases; organic diseases of the heart, 10 cases.

In this paper I propose to deal only with the first two sections of cases (and that from the point of view of clinical observation), to consider the questions of pathological causation, and briefly to indicate the treatment which has seemed to be the most efficacious.

I. *Pain referred to the Heart.*

In a short communication published in the 'Lancet' of January 2nd, 1892 (p. 14), I gave a summary of the cases I had observed until that date in which, after attacks of influenza, pain had been referred by the patients closely to the region of the heart. The pain was in some instances paroxysmal, in others more or less constant. In certain of the cases manifesting paroxysmal pain the symptoms closely approached those of angina pectoris. In a typical athlete, five months after influenza there occurred attacks of intense præcordial pain, as of a "grip" or "screw," which in a moment caused the patient to fall prostrate and, for a brief period, completely unconscious. In a lady aged forty-one, attacks of intense pain, localised in the second left intercostal space—where was a tender spot—were initiated by moderate exertion. In a gentleman aged thirty-three, paroxysms of pain at the heart of much less severity than those in the former instances occurred at intervals ever since an attack of influenza, and in these seizures the patient had the feeling of impending death. Such feeling was graphically described by another patient "as if his heart were a pendulum suddenly stopped at one swing." In others the pain was long persistent; in three cases it was described as a dull aching referred to the area occupied by the heart, accompanied in one case by a tingling felt down the left arm.

In the series of cases that I am now about to review pain

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was referred to the heart region in twenty-three instances. Of these the pain was of *paroxysmal* character in six. In a lady aged twenty-six, intense pain at the heart occurred one week after an attack of influenza; the attacks were many times repeated, and, according to reliable evidence, were attended with loss of consciousness. In a gentleman aged fifty-four, sudden and intense pain at the heart occurred after a second attack of influenza; the pain was also felt down the left arm, the neck, and the back. A feeling of deadness down the left arm was experienced in one case; and in another, at the time of the attacks, which were very severe, pain was felt in the throat. That the loss of consciousness observed in some of the cases could be independent of the severity of the pain was shown in the case of a gentleman aged twenty-three, in whom a "dead pain" at the heart was noted, but who lost consciousness on two occasions. There were no convulsions nor epileptiform symptoms.

Although the phenomena of angina pectoris were closely simulated in some of these cases, I met with none in which there was good evidence of an abnormal tension within the arteries, either at the time of the paroxysms or during the intervals. In all the cases there was nothing to show any change in the conditions of the myocardium. In those which manifested the most intense paroxysms, the conditions of the heart both in rhythm and in integrity of structure, so far as could be determined by physical examination, were quite normal. None of the cases were fatal.

With regard to the *sites* of pain, the patients referred the suffering to certain portions of the area occupied by the heart itself in nine instances, and to the sternum in eight. In a few cases the areas in which pain was felt could be mapped out with considerable precision, and these are indicated in the chart.

In most cases the constant (as distinguished from the paroxysmal) pain was of a dull aching character, but subject to exacerbations. The parasternal pain was exceptionally as of a knife cutting; in a small minority a

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tender spot was found close to the sternal border. The pain extended down the left arm in three instances, in one of these the disturbed sensation being described as a

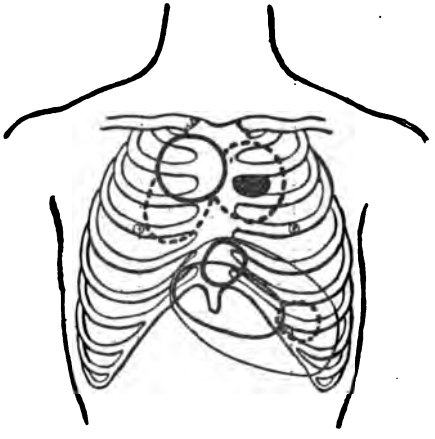

Figure indicating the positions of pain referred to the heart in eight cases.

deadness ; in another case it was severely felt in the back. In many cases the suffering was so precisely felt in relation to the heart, that the patients could not be convinced that there was no organic disease. The pulsations were accompanied by pain and by most uncomfortable sensations, though the rhythm was normal and tranquil, and the heart-sounds well pronounced and in their due relations one to the other.

As to the pathology of the painful affections referred to the heart in sequence to attacks of influenza, I said in my former essay that it seemed to me probable that though the cardiac crises during the acute stage of the disease might be due to involvements of the nerve elements in the bulb, the remote symptoms should be ascribed to a neuritis affecting the sympathetic ganglia, the vagus, and the sensori-motor nerve-trunks.

Abundant evidence has accumulated to prove that multiple neuritis may occur at various periods after attacks of influenza. I have observed several cases in which there

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has been distinct clinical evidence of neuritis affecting the brachial plexus, and in some of those in the category we are now considering numbness and tingling were manifested for long periods in the forearm and fingers (see also cases by Remak ('Berlin. klin. Wochenschr.,' February 24th, 1890), Henoch (ibid.), Althaus ('On Influenza,' p. 187, *et seq.*), Church ('Journal of American Medical Association,' November 1st, 1891), Westphalen ('Petersburg. med. Wochenschr.,' 1890, p. 48), Draper ('New York Medical Record,' 1891, p. 9), &c.).

I have said in the paper to which I have made allusion that it is probable that the ganglia of the sympathetic may in some cases be involved in inflammatory changes. This has been proved in the case of the ganglia within the abdomen by Ferguson ('Alienist and Neurologist,' October, 1890), who found in a case which, after an attack of influenza, manifested agonising pain in the abdomen, and ended fatally, inflammatory exudation about the ganglia and degeneration of some of the nerve elements. I have not met with any instance in which there has been post-mortem evidence of neuritis affecting the cardiac ganglia, but there seems reasonable probability that in some of the cases manifesting agonising pain in the situation of the cardiac plexuses this might have occurred. In most cases there might be a greater probability of a visceral neuralgia, analogous to that of the pleuræ or of the stomach and intestines, and due, perhaps, to a disturbance of the nerve-cells within the cerebro-spinal axis. That there may be a disorder of the vagus at its origin or in its course is rendered very probable by some of the symptoms observed, for example those of a sense of impending death, of arrest of the heart's action, and associated dyspnœa.

In the majority of cases the pain at the heart began to be manifested from one to four months after the attack of influenza, but some came under observation at much later periods, and it was clear that such pain could be experienced at least eighteen months after the initial disease.

In the treatment of the cases manifesting agonising

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paroxysms rest was of course of paramount importance. Neither inhalations of nitrite of amyl nor the administration of nitro-glycerine seemed to give any satisfactory results. In the case of the most acute suffering, the attacks causing syncope, a course of bromide and iodide of sodium with aromatic spirit of ammonia was prescribed, and at the time of the paroxysms a draught containing pure ether (ℿv) and tinctures of lavender and sumbul (of each ℿxx) in chloroform water (℥j). Subsequently five grains of sulphate of quinine were given daily—once in the twenty-four hours. The attacks diminished in severity, and at the end of four months' treatment all pains had ceased. The patient has written to me stating that at rare intervals he had pain, though of slight intensity, after omitting the treatment, but now he is in perfect health. In other cases of severe pain the hypodermic injection of hydrochlorate of morphine (gr. ¼ to gr. ⅓) was employed; but in order to obviate, if possible, any tendency to syncope, diffusible stimulants (ether, ammonia, valerian, or sumbul) were administered at the time of the injection. As a general rule opiates were not well borne.

In cases of moderate severity, counter-irritation of the præcordia by mustard poultices sprinkled with tinctures of opium and belladonna gave relief. I also used local applications of a hot solution of salicylate of sodium, a piece of lint being soaked in a saturated solution of the salt mixed with an equal bulk of hot water, applied to the præcordia, and covered with a piece of lint soaked in olive oil, surrounded by cotton wool. I was convinced of the great comfort afforded by this method of treatment.

In some instances of long-continued pain I advised, as in cases hereafter to be noted, the use of the continuous galvanic current. A gentleman aged sixty-two, after two attacks of influenza, suffered severe pain of a protracted character localised in two areas, one about the apex of the heart (and especially the right ventricle), and the other a defined circle with its centre at the aortic cartilage. The pain was aggravated by any exertion. The ordinary medi-

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cinal methods of treatment having after two months failed to give decided relief, I advised the use of a continuous current from four cells from the nape of the neck to the course of the vagus in front of the neck, administered for six minutes three times a day. There followed distinct though gradual improvement, and in one month the pain entirely ceased. Afterwards the patient expressed himself as being in perfect health.

II. *Morbid Acceleration of the Heart's Action (Tachycardia).*

I have considered as examples of a morbidly rapid heart's action those in whom the observed rate, which according to the evidence persisted for protracted periods, was, or exceeded, 100 per minute. In thirty-two cases the rate was from 100 to 120; in five it was above 120, the maximum being 128. The rapidity of the heart's action was maintained in the majority without any feelings of subjective discomfort on the part of the patient. In only nine cases were there paroxysmal palpitations; two of these manifested muscular tremors. In a considerable number of cases the already rapid heart was made yet more rapid by even slight movements on the part of the patient, and not infrequently there were periodic accelerations without assignable cause. The mere change of position from sitting to standing caused an increased frequency of pulse, of 40 in two cases, and of from 12 to 24 in six others. The lifting of the left arm increased the pulse-rate from 100 to 120 in one case. Several cases presented great variations, and the varying rate could be noted during the auscultation of the heart. In the case of a female aged thirty-five, in whom a pulse of 120 was generally observed during the day, it was said that the pulse became slower than normal during the night. In the case of a lad of eighteen, the pulse, which at an early period was slow (54), became habitually quick (108 to 120) later.

The quickened action of the heart was in very few

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cases attended with subjective discomfort. The converse might be the case. For example, a gentleman who for a long period had a pulse-rate of about 112 per minute without cardiac distress came to me complaining of much pain in the heart region, and the pulse was regular, the rate 80 per minute, and the heart-sounds normal. I have noticed the coincidence of signs of pain with abnormal infrequency of pulse (bradycardia).

Many of the cases of post-influenzal tachycardia presented also other of the *signs of Graves' disease*. A girl of thirteen, just convalescent from influenza, manifested an increasing enlargement of the thyroid, with throbbing in its situation and a feeling of "lump in the throat." The enlargement was of both lobes, chiefly the right; much heaving pulsation was felt over the gland, and murmurs were loudly heard in its situation. The pulse-rate was noted as 100 to 120, and palpitations were severe. The additional signs were tremors, perspirations, and some abnormal pigmentations of the skin. There were, however, no lid signs nor exophthalmos.

In a second case (of a lady aged twenty-two) palpitations began to be noticed about eight months after an attack of influenza. The pulse-rate was generally about 120, sometimes 160. The right lobe of the thyroid was enlarged.

In other cases the *exophthalmos and the eyelid phenomena* of Graves' disease were observed. A lady aged fifty-nine manifested subsequently to influenza a pulse-rate of about 128; there was increasing exophthalmos, and some retraction of the upper eyelid (Stellwag's sign) was noticed, but no von Graefe's sign.

In a gentleman aged fifty-four, after a second attack of influenza, the pulse-rate was noted to vary between 96 and 112; there were sudden and severe pains referred to the heart region, the neck, and the left arm; outbreaks of flatulence also occurred. There was no thyroid enlargement, but both Stellwag's and von Graefe's eyelid signs were manifested.

In a third case (a lady aged fifty-one) the pulse varied

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between 104 and 128, the heart was made uncomfortable by any exertion, there was some muscular tremor, and distinct exophthalmos was noted.

In two other cases of tachycardia (in males) Stellwag's lid sign was noted. In a lady aged thirty-one both Stellwag's and von Graefe's signs were present, there were gastric disturbances, and abnormal pulsation was felt within the abdomen.

Phenomena frequently associated with Graves' disease were observed in many cases. Palpitations, flutterings, flushings, perspirations, and faintnesses were noted in seven instances; pulsations were experienced in the carotids and subclavians, in the abdominal aorta, and in the splenic artery in three others. Systolic murmurs, considered to be independent of organic disease, were found over the pulmonary artery in four and over the aorta in two cases. Paroxysmal attacks of dyspepsia (flatulence, nausea, gastralgia, and diarrhœa) accompanied by dyspnœa, attacks which I have termed "vagus storms," occurred in five instances.

I cannot doubt, from the evidence of the cases which I have observed, that Graves' disease can be brought about by influenza. Although I have not met with a case in which the complete "ensemble" of phenomena has presented itself, it is clear that so many of the signs have concurred in some cases, and those which have been missing in some have been evident in others, that I cannot doubt that the condition of disease produced is identical with that of exophthalmic goître.

Colley ("Ueber Morbus Basedowii nach Influenza," 'Deutsche medizinische Wochenschr.,' August 28, 1890) described the case of a woman in whom exophthalmos occurred during an attack of influenza, and was followed by bilateral bronchocele and by tachycardia (pulse 150 to 180). In this case, therefore, all the cardinal phenomena were initiated (cf. Althaus, 'On Influenza,' London, Longmans, 1892, p. 199).

As to the question of the mode of pathogenesis of the

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condition of rapid heart in this series of cases, I do not think that there is any reliable evidence of the intervention of myocarditis, or any discoverable change in the cardiac muscle. In none were there signs of enlargement of the heart, nor of the usual phenomena of cardiac failure; in many, during the period of abnormal acceleration of the heart's action, subjective discomfort was completely absent.

I consider also that the evidence in no degree supports the theory that a morbid activity of the thyroid gland could be the cause of the tachycardia. In the great majority of the cases the thyroid seemed to be of strictly normal dimensions, or even subnormal.

In two cases presenting the associated signs of Graves' disease enlargement of the thyroid took place at an early period (perhaps the earliest) in the evolution of the symptoms, and in one of these application of ice-bags over the gland seemed to control the cardiac tumult, and to contribute in no small degree to the recovery of the patient. On the other hand, in the bulk of the cases—even of those manifesting, besides the tachycardia, many of the associated symptoms of Graves' disease—there was no evidence whatever of enlargement of the thyroid; and in many the gland was quite undetectable, so that the view might be advanced that it was even smaller than normal.

The series of cases of post-influenzal tachycardia would seem to me to rank in all respects with those of rapid heart from various causes, of which I have made a previous study ('Transactions of the Medical Society of London,' 1890, p. 467, *et seq.*); and I submit that the conclusions which I deduced from the evidence afforded by the latter can be in like manner legitimately derived from the former. These conclusions are that the ætiological factor in cases of tachycardia, when there is sufficient evidence to establish any, is overstrain, physical or mental; that such tachycardia may continue for long periods as the chief or only notable morbid pheno-

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menon, or may be associated in greater or less degree with the other well-known phenomena of the completely developed Graves' disease; that it is only in a small minority that any enlargement of the thyroid is observed at the earliest period of the affection; that in a larger proportion such enlargement is to be detected at later periods of the evolution of symptoms; and that in a majority there is no evidence of an abnormal condition of the thyroid at any stage.

I submit, therefore, that the effect of influenza in those cases which present a morbid acceleration of the action of the heart is in strict accord with that of a sudden and severe shock to the nervous system. As to the proximate mode by which such morbid acceleration may be brought about, we may entertain the following hypotheses: there may be an inhibition of the action of the vagus, an over-action of the accelerator nerve-mechanism of the heart, or those two conditions in varying degrees combined.

It is well established that disease encroaching upon the trunk of the vagus or involving its fibres may induce abnormal rapidity of the heart's action. This may occur from the pressure of tubercular glands, as in a case recorded by Merklen, in which the right pneumogastric was compressed and the pulse-rate went up to 156 (cf. Huchard, "L'Asystolie nerveuse," 'Rev. gén. de Clinique et de Thérapeutique,' 27 Septembre, 1893, p. 612). A like result has followed a compression of the vagus by thickened pericardium. Neuritis involving the fibres of the pneumogastric has produced the same symptom. Besançon records the case of a phthisical subject, aged thirty-three, in whom tachycardia became manifested; death occurred from heart failure, and neuritis of the left pneumogastric was demonstrated at the autopsy, the inflammation evidently spreading from the tubercular lesion. Instances of polyneuritis involving the pneumogastrics and inducing tachycardia as a symptom have been recorded by many observers as the result of alcoholic poisoning, and in sequence to many of the infectious diseases. A consider-

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able number of such cases have been observed after influenza.

I have had under my care a female patient who suffered from an attack of influenza just previously to admission into the wards of the London Hospital for paralysis of the lower limbs. There were the typical dropped feet and other signs of alcoholic neuritis. There was a decided history of alcoholism. During my observation I noted the manifestation of quickened breathing, but there was not the slightest indication during respiration of any movement of the diaphragm. There was evidence of paralysis through the phrenics. Quite suddenly the pulse became rapid, so persisting for several days, and then coming down to the normal. I concluded that there was a temporary neuritis of the vagus. This is one of several cases under my care in which I came to the conclusion that influenza reinforced the morbid phenomena of alcoholic neuritis.

From these data I consider it very probable that post-influenzal tachycardia is due to a disturbance of the vagus, at its origin or at some part of its course, whereby its controlling power, as transmitted by its cardiac branches, is in more or less complete abeyance.

It may yet be considered whether an irritant lesion of the sympathetic may produce the symptom. It has been shown that Graves' disease may be initiated by influenza, and I have given evidence to render it probable (or even certain) that neuritis of the sympathetic ganglia may occur in the latter disease. Inflammatory changes have been demonstrated in their midst in Graves' disease. Dr. W. S. Greenfield found in the cervical sympathetic (especially the middle cervical ganglion) very marked changes indicating that the ganglia may become the seat of a subacute inflammatory process ('British Medical Journal,' December 9th, 1893, p. 1266). Drs. Cruise, Reith, Shingleton Smith, Virchow, Lancereaux, and Geigel had long previously recorded the organic changes which had been observed by them in fatal cases of

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Graves' disease—changes indicating either inflammatory infiltrations or sclerous transformations. In Dr. Greenfield's cases, however, there were also morbid appearances in the central nervous system; these are stated to have been "closely analogous to those seen in the sympathetic ganglia," and, in addition, scattered hæmorrhages whose sites were the pons and medulla, and in one case the floor of the fourth ventricle. Dr. Hale White ('British Medical Journal,' March 30th, 1889) had previously observed like hæmorrhagic extravasations in a case after death from Graves' disease in the same situation, obviously close to the nuclei of origin of the vagus.

It seems probable that though the lesions of the sympathetic might be capable of inducing the tachycardia, these can only be regarded as concurring or reinforcing causes. It is scarcely likely that the inflammatory conditions capable of producing irritation should last for months or even years; whilst if sclerosis or calcareous degeneration could be invoked as a cause, the complete recovery which occurs in a large number of the cases would be improbable. It appears to me that a lesion impairing the power of the vagus, often slow in recovery, should be regarded as the chief cause, a neuritis of the sympathetic being in some cases a concurring factor. It is possible that the tract of the cardiac accelerator nerves within the spinal cord may in some cases be affected by an irritant lesion.

Though improbable as a chief cause, excessive secretion by the thyroid, the result of subinflammatory changes in the gland, may be looked upon also as in some cases a concurring agency.

In the treatment of patients manifesting post-influenzal tachycardia I have found the administration of digitalis, belladonna, and all the usual heart tonics ineffectual if not even harmful. This accords with my experience of tachycardia from all causes when unaccompanied by structural disease. M. Huchard states that digitalis has no power of control over morbid acceleration of the heart's action when

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this is dependent on disturbance of the nervous mechanism, and not on organic disease: with this statement I entirely concur.

The bromides of potassium, sodium, or ammonium in moderate doses (twenty grains thrice daily) have been useful in some cases, but if persisted in for long periods they increase the lassitude and general enfeeblement. It is well to omit them for two or three days at the end of each week. In cases of insomnia and of restlessness at night it has seemed to me that chloralamide in doses of twenty or twenty-five grains is the safest and best agent. In some cases, however, it is inefficient, and in such sulphonal in twenty or twenty-five grain doses often procures the required sleep. This should not be administered every night, however, for some patients manifest a craving for the drug, and it may have some toxic action. In a small minority morphine (best administered subcutaneously) is a necessity, but it should be used with caution.

In the cases in which dyspepsia is a marked symptom—and it is so both as concomitant and result of vagus storms—alkalies with pancreatin have been administered with advantage. In some cases arsenic, given in the form of Fowler's solution in small (two-minim) doses, gradually increased to five or seven, has been very beneficial. Iron preparations have generally been ill borne.

Medicinal treatment alone has been, according to my experience, very unsatisfactory in the treatment of tachycardia. There may be relief of some of the symptoms and an improvement of the general condition, but the morbid acceleration of the heart's action continues uninfluenced.

I consider, however, that there is good evidence that much may be done by the use of the continuous galvanic current. I began to treat cases of Graves' disease by weak galvanic currents more than twenty years ago—in 1873. At that time the rheophores were placed over the spinal column in the cervical and dorsal regions. More recently they were arranged so that the current passed from the cervical spine to the region of the vagus in the

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neck—the anode, a flat plate covered by moistened lint, being closely applied to the nape of the neck just above the seventh cervical vertebra; and the kathode, a button similarly covered and moistened, gently pressed in the groove between the thyroid cartilage and the sterno-mastoid muscle, and moved slowly downwards outside the trachea to the clavicle. The current should be weak—from two to four milliampères, such as may be obtained from four to six ordinary Leclanché cells. It is almost imperceptible to the patient, though I have known a sensitive lady who could not bear a current from more than two cells. It cannot be doubted that by this proceeding the medulla oblongata and base of the brain, as well as the trunk of the vagus, are in some degree influenced. It is the consensus of a considerable number of observers that the rate of the heart's contractions can thus be notably reduced. Eulenberg and Schmidt observed that the pulsations of the normal heart could be retarded by four to sixteen per minute, and that in pathological accelerations the reduction was more considerable. Beard and Rockwell found generally a diminution of two, three, or four beats in a minute, but in some cases there was an acceleration. I may parenthetically note that the currents which they employed were too strong. Damion has given many cases in which pathological acceleration was reduced and cure effected (Althaus, 'A Treatise on Medical Electricity,' 3rd ed., p. 165; Beard and Rockwell, 'A Practical Treatise on the Medical and Surgical Uses of Electricity,' London, H. K. Lewis, 1889, p. 620; 'L'Électro-thérapie,' January, 1889, p. 27).

Full details as to the employment of the continuous galvanic current in cases of Graves' disease have been given in a very practical paper by the late Mr. H. W. D. Cardew ('Lancet,' July 4th and 11th, 1891, pp. 6 and 64). My late friend and I worked together in our observations of many cases, and the communication embodies the conclusions of both. Suffice it to say here that we discarded the interrupted current as used by some Continental

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physicians, and found our best results from the weak continuous currents.

The value of the treatment becomes practically "nil" unless it can be applied by unskilled hands, for it should be employed at least three times a day for periods of from six to ten minutes. Much improvement must not be expected in less than four or six months. It is preferable, of course, that an expert should practically instruct the patient at the outset how to use the battery; but the proceeding is so simple that this is not essential. The usual medical attendant can always arrange this, and see that the poles are properly applied. He should caution the patient against carelessly short-circuiting the battery by allowing the poles to communicate when not in use.

A battery of four to six cells is very inexpensive, and can be made very portable. This slight expense to poor patients can be very much reduced by the use of the ordinary cheap Leclanché cells, properly coupled and furnished with covered wires and terminal rheophores, which can be bought for very little.

The following are brief notes of cases thus treated.

Case 1.—A girl æt. 13 suffered an attack of influenza in January, 1892. There were severe pains, chiefly referred to the muscles, and the patient was in bed or in the house a whole month. She came under my notice in April following. I noted much enlargement of both lobes of the thyroid, especially the right; there was much throbbing, sometimes painful, of the arteries of the neck. The pulse was 120; occasionally there were severe palpitations. A loud systolic murmur was heard over the pulmonary artery, and a continuous murmur with systolic reinforcement in the neck. Some frecklings and maculations were seen upon the face. There were muscular tremors and occasionally profuse perspirations. Ordered continuous current (from four cells) from nape of neck to course of vagus in the neck, and to thyroid swellings for

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six minutes three times a day; ice-bags to be applied at intervals over the thyroid.

After one month there was slight improvement in the symptoms, the pulse-rate being generally 100, but palpitations were frequent. The treatment by galvanism (and the ice-bags occasionally) was continued, and ten-grain doses of sodium bromide with two minims of Fowler's solution were given in a mixture three times a day. Further improvement followed, till in October a second attack of influenza occurred, and the treatment was interrupted for about a fortnight. Then the thyroid swellings had again increased. The galvanism and the application of ice-bags were resumed. When I saw the patient in November the pulse-rate was 92; there were no tremors nor perspirations. In January, 1893, after nine months of treatment, there was almost recovery. The pulse was 88 per minute; there were no palpitations, the thyroid swellings were much reduced, and there were no throbbings in the neck. I have heard since that there has been a restoration to good health.

Case 2.—A lady aged 54 had an attack of influenza in January, 1892, and subsequently suffered from dyspepsia, uncomfortable sensations referred to the throat, palpitations, and insomnia. She was seen by me in October, 1892, when I found enlargement of the thyroid, especially of the right lobe; the pulse 100 per minute, occasionally intermittent; palpitations were said to occur frequently; there was almost constant nausea, and headaches were distressing. After one month's treatment by bromides there seemed to be no improvement, and galvanism was commenced as in the former case. After one month of the treatment by galvanism there was much less palpitation, and scarcely any nausea was experienced. The pulse-rate was 92 per minute. Then, in January, 1893, sudden epigastric pain occurred, and all the symptoms became aggravated; the pulse 120, with palpitations. The galvanic treatment was continued. At the end of February

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there were no attacks of indigestion nor palpitations; the pulse-rate 96 to 100, the thyroid swelling much diminished.

In April (after five months of treatment by galvanism) pulse 76 to 80; palpitations at very rare intervals. Very little subjective discomfort at any time.

It is, of course, a legitimate question whether the good results in these cases were due to the galvanism only. It may be urged that the cases might have recovered in the periods mentioned without the treatment. I think that most observers who have seen much of Graves' disease will think this very unlikely. The influence of predominant idea—of suggestion—may be invoked as a probable explanation of the recoveries by some critics. I must record my own conviction that the use of the galvanic current in the manner I have indicated was a sure although a slow agency for good.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. vi, p. 121.)

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A CASE OF ACANTHOSIS NIGRICANS.

BY

MALCOLM MORRIS, F.R.C.S.Ed.

Received May 28th—Read June 13th, 1894.

The following case is an example of one of the rarest of skin diseases, and is, as far as I am aware, the first of the kind reported in this country. The patient, who was sent to me by Dr. Mackern, of Blackheath, is a single woman æt. 35, and was admitted into St. Mary's Hospital under my care on February 15th, 1894. The notes have been condensed from the report made by my clinical assistant, Mr. Randolph Grosvenor. There is nothing in the patient's family history that would seem to have any bearing on her present condition. She says she had typhus and scarlet fever in childhood; otherwise her health was satisfactory up to the time she was attacked by the disorder from which she is now suffering. For more than a year before that, however, her menstrual history shows great irregularity. In the autumn of 1892 she began to suffer from menorrhagia. This continued for nearly a year, after which the periods, while continuing to be unduly frequent (once a week), were scanty in amount. Since April, 1893, she has suffered from leucorrhœa. About August, 1892, her hair, which had pre-

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viously been thick and long, became dry and wiry, and began to fall out. She had always been subject to dryness of the hands and feet, but her skin generally was soft in texture before the beginning of the present condition. There seems to have been no marked tendency to the formation of warts. About five years ago there were three warts on one of her thumbs, but these had disappeared before she came under observation. One pedunculated wart had, however, been present in each axilla for several years. She has always been of an excitable temperament, and the neurotic element in her constitution has become much more pronounced during the development of the skin affection.

The first appearance of the lesions dates from some eight months back. In the beginning of October, 1893, she noticed a general bronzing of the skin over the upper half of her body. About the same time crops of warts came out on the backs of her hands, the nails became uneven, and the skin between the fingers rough. Patches of skin on the chest, sides, and to a less extent on the back, became harsh and blackish. In the axillæ the harshness soon passed into a wart-like thickening, the hypertrophied skin being quite black. At the end of December the skin on the palms and flexor surfaces of the fingers became thickened, and of an orange-yellow colour, and fissures formed on the finger ends. At the beginning of February the warty condition of the axillæ had greatly increased, and deep cracks had formed in the hypertrophic mass, from which a discharge exuded.

In the first week of January she noticed that her underclothing was stuck to her abdomen by a thick discharge; the source of this was found to be the umbilicus, which looked red in the middle. A week later the patient noticed a growth in that situation, which increased in size somewhat rapidly for two or three weeks, and then seemed to come to a standstill. The discharge was purulent in character, and was particularly abundant on movement.

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The first sign of involvement of the *mucous membranes* appears to have been a peculiar dryness of the lips, which the patient first noticed at the beginning of December, 1893. In the middle of January "lumps" formed on the tongue, and a few days later the mucous membranes on the inside of the cheeks became rough and wrinkled. At the end of the same month warts were observed on the roof of the mouth. Shortly before the patient's admission she noticed that the inside of her ears felt moist, and emitted a disagreeable smell, but there was no actual discharge; there was also some impairment of hearing. The condition became steadily worse up to the time of her admission, fresh crops of warts appearing in various parts of the body, the black patches increasing in size, and the bronzing becoming more diffused.

Condition on admission.—The patient looks wasted and her muscles are flabby, but her general health is fairly good. The temperature is normal. She is nervous but not hysterical; the knee-jerk is almost entirely absent on both sides.

Skin: The skin lesions present three principal features: 1st, diffuse brownish discoloration or bronzing; 2nd, patches of inky blackness; 3rd, warty growths. It will be most convenient to describe the appearances as seen on examining the patient from the head downwards. The skin of the whole upper part of the body is distinctly bronzed. The face looks slightly swollen and flushed, the redness deepening when she is spoken to. The skin of the face is dry and scurfy, and there are small warts on the skin and forehead, chin and cheek. The meatus auditorius on either side is packed with warts which obstruct the passage. The skin of the neck both in front and behind, and on the under surface of the chin, is coarse in appearance and stained black all round; at the back of the neck the discoloration extends upwards for a short way on the scalp, and in front it runs downwards from the neck in two narrow strips on either side of the middle line to the chest. The whole of the skin of the

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neck is rough and the natural folds are exaggerated. The roughness extends over the front of the chest, sparing the breasts except the left, which is very slightly affected; on the right mamma the skin is normal, but is dotted here and there with a few small warts. Both nipples are slightly discoloured and warty. Between the breasts the skin is coarse and blackened; on the sides of the chest it is fairly normal, with the exception of a small discoloured area immediately below the axillæ. In the axilla on each side the disease is seen at its highest degree of development. The whole of the space is occupied by black masses of warty growths separated by deep furrows, from which oozes a small quantity of offensive discharge. At one time the whole of the hypertrophied mass was quite black, but at present there is in the middle of each axilla a raw-looking patch of a bright flesh-colour. On the upper limbs the skin, except at the bend of the elbow, is normal down to the lower third of the forearm, but a number of small warts are scattered about, especially at the flexor surfaces. At the bend of the elbow there is a deep brown stain dotted with black spots. The natural folds of the skin are exaggerated. On the extensor surfaces nothing but slight roughness is observed. The palms of the hands and the flexor surfaces of the fingers are greatly thickened and show very marked deepening of the natural lines. The whole of the skin in these situations is of a light orange-colour, and feels like rough piled velvet. The backs of the hands as low as the knuckles and the backs of the wrists are fairly normal though rough, but at the edges and on the backs of the fingers the condition of the skin approximates to that of the palms.

Over the whole of the abdomen the skin is bronzed, rough, and wrinkled. In the middle line in front, a black band continuous with that between the breasts spreads downwards for some inches. In the situation of the umbilicus there is a button-like growth of considerable size, clearly papillomatous in nature. It is circular

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in shape, measuring 1½ inches across and about ½ inch from above downwards; it is slightly narrowed at its attachment to the surrounding skin, and is surrounded by a blackened zone one inch in width. From about three inches below the umbilicus to the groins the skin presents no anomaly beyond slight general bronzing. On the fronts and inner aspects of the thighs the skin is natural, but in the latter situation a few small warts are scattered about. Behind towards the gluteo-femoral fold there is thickening with brown discoloration. Over the patellæ and behind the knees the skin is also thickened and generally discoloured, with here and there patches of blacker staining, especially in the popliteal spaces. From the knees down to the ankle the skin is dry and wrinkled, but otherwise normal. About the ankles in front and behind there is some discoloration and creasing. The feet are normal on the dorsal aspects, but on the sides and the soles the skin is dry, rough, and furrowed; the skin of the heel and of those parts of the foot which touch the ground in walking is very rough and thick, and of a yellow-brown colour. The skin on the ends of the toes is also very rough and thickened, and the nails are pushed some way out of their beds by a mass of horny tissue.

Mucous membranes.—Nearly all the visible mucous membranes are affected. The lips are swollen, dry, and of a pale orange-colour. The soft palate is covered with masses of small warty growths; and a similar condition is seen on the anterior portion of the hard palate on either side, on the cheeks, on the tongue, and on the gums. The throat is unaffected. The mucous lining of the vagina is covered with growths of similar appearance. The patient suffers from leucorrhœa, but the vaginal discharge appears to have been noticed some time before the onset of the skin affection.

It will thus be seen that the principal objective features of this remarkable disease are discoloration and wartiness of the skin. The distribution of the black stains is fairly symmetrical, and they show a marked predilection for

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the flexures and large natural folds of the integument (axillæ, bends of elbows, behind the knees, neck, between the breasts, &c.). On close inspection the black patches are seen to be caused by numerous black granules. The individual granules as a rule are of about the size of a pin's head, but in the places where the stain is deepest (as in the axillæ) they are somewhat larger. They appear to be situated in the superficial layers of the epidermis, and can be readily brought to the surface by pinching up a fold of skin and rolling it between the fingers. The warty growths are simply papillomatous in structure as well as in appearance. There would seem to be no constant relation between them and the staining, for although the circumference of the warty masses in the axillæ is blackened as if by gunpowder, the warts on the face, arms, and mucous membranes show no trace of blackening. On the other hand, many of the black patches, as on the neck and in the bends of the elbows, though rough on the surface, are not warty.

Beyond the discomfort caused by the condition of the lips and mouth there are no subjective symptoms. The patient when first admitted complained of pain about the pubic region and the thighs, but this was probably connected with disordered menstruation.

The urine presents no abnormality.

While the patient has been under observation the black staining has spread over the entire surface of the abdomen and the whole of the back as high as the middle of the scapula. A few warts have developed on the scalp and in the lumbar region. About the middle of March it was noticed that a thick growth of white hair was taking place on the face, and to a less extent on the chest and abdomen. So luxuriant did the growth become on the face, invading the forehead and even the nose, that the patient asked to be allowed to shave. The hair on the head is rapidly turning grey, and is becoming very thin. On the other hand, there has been some improvement in other parts, especially the hands, feet, and knees; in these

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situations there is less thickening and the skin is softer.

On March 15th the umbilical wart was removed; nothing but skin was involved in the growth. The warts in the auditory meatus were for some time treated by the application of salicylic acid, with the result that the hearing greatly improved. In other respects the treatment has been purely expectant.

I think there is no doubt that this case is an example of the disease to which Unna has given the name of *Acanthosis nigricans*, the substantive denoting a member of Auspitz's group of Acanthomata (ἄκανθα, prickle), and the adjective expressing the most striking feature of the affection. The symptomatology of the disease may be summed up as horny thickening with discoloration and wartiness of the skin. Only two cases have so far been recorded, one by S. Pollitzer [1] and the other by V. Janovsky.[2] Pollitzer's patient was a woman aged sixty-two, in whom the upper extremities, the neck, the mouth, part of the trunk, and the genito-crural regions were all attacked about the same time. The skin became discoloured ("dirty brownish"), rough, and covered in places with "diffuse warty prominences," the natural folds and furrows on the palm of the hand and elsewhere being strongly marked. The hard palate, tongue, and gums were covered with "fine granulations resembling small venereal warts, but softer to the touch." The patient recovered (apparently independently of the treatment pursued) in a few months as far as the condition of the skin was concerned, but soon died of anuria, which was believed by her medical advisers to be due to an internal cancer. No *post-mortem* examination was permitted. Microscopic sections of some of the warts showed hypertrophy of the Malpighian layer, the rete presenting, instead of the normal regular arrangement of its cells in rows or parallel layers, a confused

[1] 'Internat. Atlas f. Selten Hautkrankheiten.,' No. x.
[2] Ibid., No. xi.

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appearance of whorls and gyri as if their atypical growth had proceeded concentrically from numerous points. There were frequent suggestions of "epithelial pearls." Careful search was made for micro-organisms, but none of a specific character were found.

Janovsky's patient was a man aged forty-two, who had for eighteen years been employed close to a furnace. The skin and mucous membranes were affected much in the same way as in Pollitzer's case. The umbilicus is described as standing out prominently in hemispherical form, darkly pigmented and papillomatous. The epiglottis and upper orifice of the larynx as well as the mouth and tongue were the seat of papillary outgrowths. The man's general health was unaffected.

Crocker has reported a case of "general bronzing without constitutional symptoms,"[1] which presents considerable analogy with those of Pollitzer and Janovsky, but the mucous membranes and the hands were not involved, and there was no thickening of the horny layer to speak of. The affection arrived at its full development within a few days, and had remained practically unchanged for eight years. Crocker thinks it was probably an effect of exposure to extreme cold.

It should be noted that while in Pollitzer's case the tongue and mouth were painful from the beginning, and in Janovsky's there was severe itching, burning, and formication in the hands and other parts, in my patient such symptoms were entirely absent.

I have no suggestion to offer as to the ætiology or pathology of this curious affection; and, indeed, it would be absurd to attempt to erect any kind of theory, of however provisional a character, on so slender a basis of clinical facts. The only indication for treatment appears to be to keep the wart formation in check as far as possible by the use of the knife in the case of the larger growths, and by the application of salicylic acid to the smaller ones.

[1] 'Clin. Soc. Trans.,' vol. xix (1881), p. 152.

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Histological report on Mr. Malcolm Morris's case of acanthosis nigricans, by J. Jackson Clarke, M.B.—The mushroom-like papillated growth at the umbilicus was attached by a pedicle of rough and harsh skin. The pedicle measured about two inches in circumference. After injecting 2 per cent. solution of cocaine the growth was removed. The skin was unusually resistent to the knife. Portions of the tissue were hardened in various media. All showed similar features, which may be expressed by the term papillomatous.

In detail the following changes may be noted :—Corium thicker than normal; the blood-vessels, veins especially, considerably dilated.

The papillary layer of the corium presented a dense small-cell infiltration at the level from which the papillæ sprang.

The papillæ themselves were large, and contained many newly formed capillaries.

The subcutaneous tissue of the growth contained greatly dilated veins, but in other respects appeared to be normal.

The epidermis showed all the features of the normal epidermis with this modification, namely, that the horny layer was thicker than normal, and in some places shreds of this layer were partly detached.

The appendages showed no noteworthy change.

No black pigment was found in the tissues of the sections examined, but a closer examination revealed within the protoplasm scattered groups of cells of the rete, bright brown granules as if of hæmatoidin.

There were very few cells undergoing mitotic [illegible] present in the epidermis, and what mitoses ther[illegible] presented quite regular forms.

The skin of the pedicle presented changes sim[illegible] but less marked than those described above.

A scraping from the darkest part of the rough sk[illegible] the front of the neck examined under the micros[illegible] showed—

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1. Epidermal scales, yellowish in colour, with hyphæ of fungi, spores, &c.

2. Many black, opaque, angular and irregular particles.

3. A few brown translucent granules, resembling amorphous hæmatoidin.

After treating and scraping in succession with boiling caustic potash, hydrochloric acid, and ferrocyanide of potassium in solution, a faint blue colour was developed. Examined with the microscope the scraping now showed the angular particles to be unchanged; most of the brown masses had disappeared. Patches of blue were visible at separate points in the field, and were probably due to the reaction of iron previously contained in the brown masses with the ferrocyanide. Only one of the black particles was the seat of the blue reaction.

Thus it is probable that the brown masses were hæmatoidin from minute intra-epidermal hæmorrhages, and it is certain that the black particles were adventitious, chiefly coal, the one of them which gave a blue reaction having probably with it a particle of pyrites.

The epidermal scales appeared to be the seat of slight diffuse pigmentation, as they were rather yellower than normal.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. vi, p. 129.)

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DESCRIPTION OF PLATES VI AND VII.

A Case of Acanthosis Nigricans (MALCOLM MORRIS).

PLATE VI.

Showing the symmetry of the eruption.

PLATE VII.

1. Acanthosis nigricans of tongue and lip.
2. Acanthosis nigricans of bend of elbow.

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M. Green. pinxt

Daniel & son & Co, chromo-lith.

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Daniels on & Co. chromo-lith.

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